



This is a digital copy of a book that was preserved for generations on library shelves before it was carefully scanned by Google as part of a project to make the world's books discoverable online.

It has survived long enough for the copyright to expire and the book to enter the public domain. A public domain book is one that was never subject to copyright or whose legal copyright term has expired. Whether a book is in the public domain may vary country to country. Public domain books are our gateways to the past, representing a wealth of history, culture and knowledge that's often difficult to discover.

Marks, notations and other marginalia present in the original volume will appear in this file - a reminder of this book's long journey from the publisher to a library and finally to you.

Usage guidelines

Google is proud to partner with libraries to digitize public domain materials and make them widely accessible. Public domain books belong to the public and we are merely their custodians. Nevertheless, this work is expensive, so in order to keep providing this resource, we have taken steps to prevent abuse by commercial parties, including placing technical restrictions on automated querying.

We also ask that you:

- + *Make non-commercial use of the files* We designed Google Book Search for use by individuals, and we request that you use these files for personal, non-commercial purposes.
- + *Refrain from automated querying* Do not send automated queries of any sort to Google's system: If you are conducting research on machine translation, optical character recognition or other areas where access to a large amount of text is helpful, please contact us. We encourage the use of public domain materials for these purposes and may be able to help.
- + *Maintain attribution* The Google "watermark" you see on each file is essential for informing people about this project and helping them find additional materials through Google Book Search. Please do not remove it.
- + *Keep it legal* Whatever your use, remember that you are responsible for ensuring that what you are doing is legal. Do not assume that just because we believe a book is in the public domain for users in the United States, that the work is also in the public domain for users in other countries. Whether a book is still in copyright varies from country to country, and we can't offer guidance on whether any specific use of any specific book is allowed. Please do not assume that a book's appearance in Google Book Search means it can be used in any manner anywhere in the world. Copyright infringement liability can be quite severe.

About Google Book Search

Google's mission is to organize the world's information and to make it universally accessible and useful. Google Book Search helps readers discover the world's books while helping authors and publishers reach new audiences. You can search through the full text of this book on the web at <http://books.google.com/>

HEART AFFECTIONS
THEIR RECOGNITION
AND TREATMENT
—
S. CALVIN SMITH

LANE MEDICAL LIBRARY STAMFORD
L681 .S66 1921
Heart affections : their recognition and



24503424761

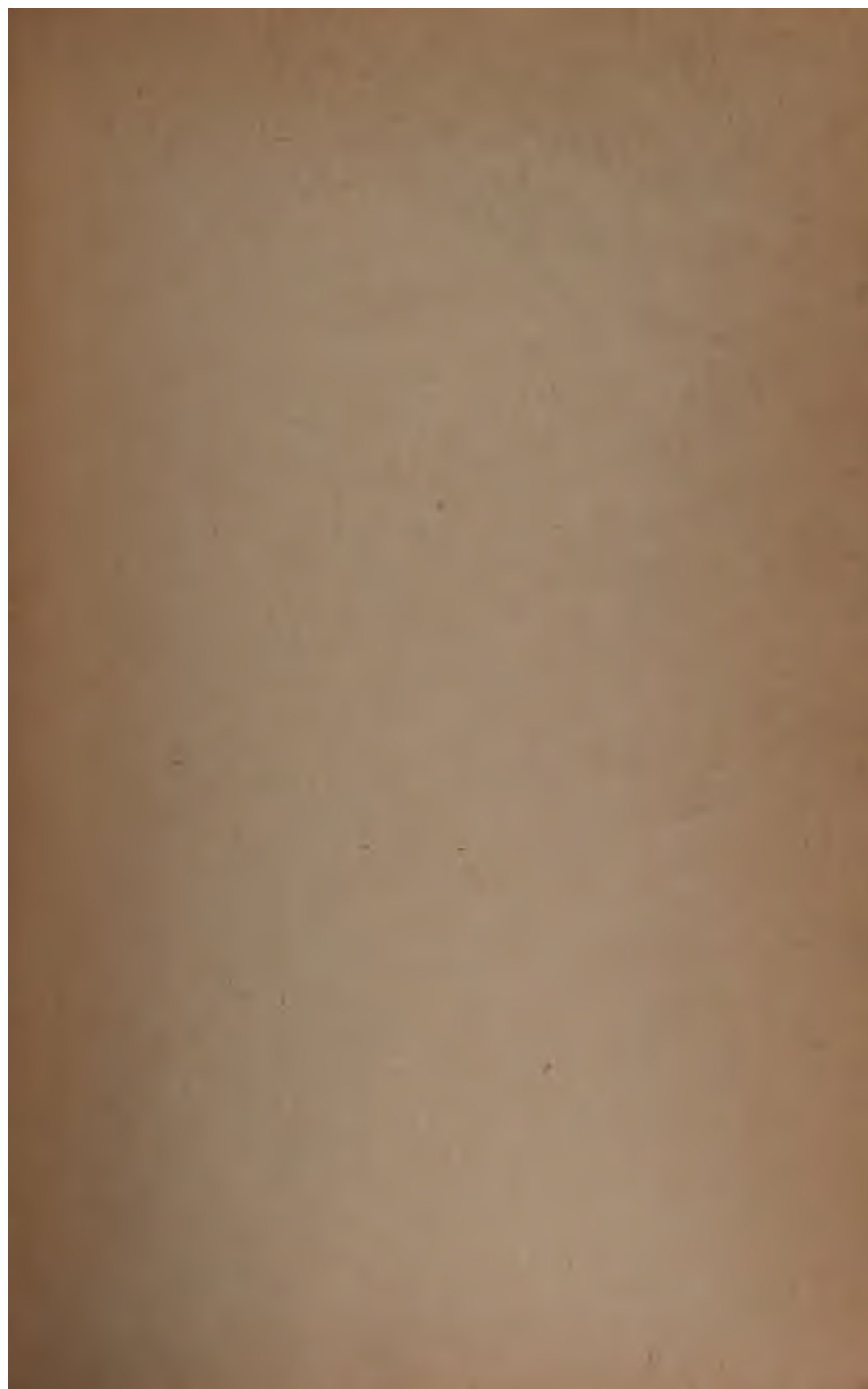
JOHN C. SPENCER, M. D.
1026 MARKET STREET
SAN FRANCISCO - CAL.

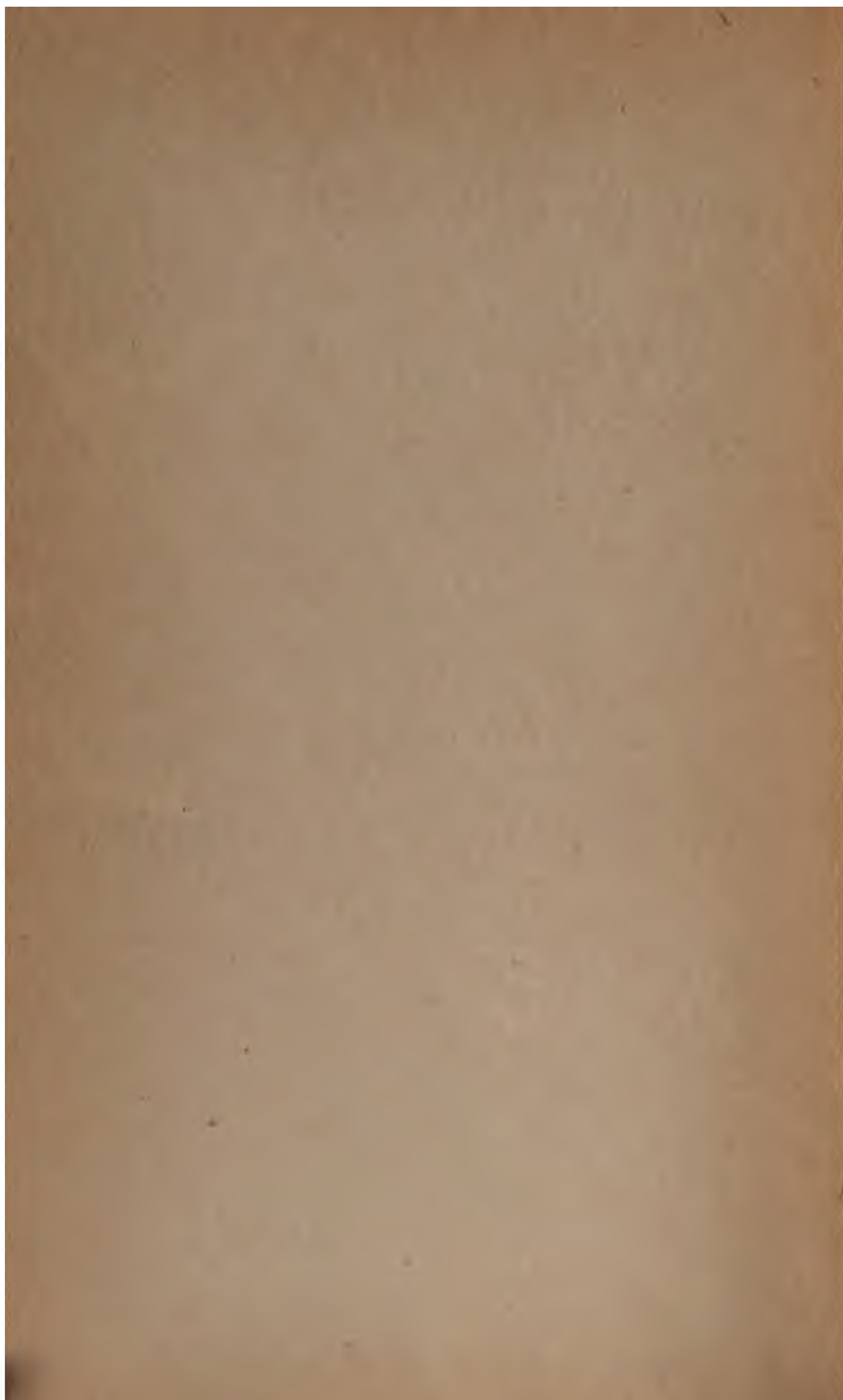


Gift of:

Dr. John C. Spencer

UNIVERSITY MICROFILMS INTL. SER.







HEART AFFECTIONS

THEIR RECOGNITION AND TREATMENT

BY

S. CALVIN SMITH, M.S., M.D.

INSTRUCTOR IN MEDICINE, UNIVERSITY OF PENNSYLVANIA GRADUATE SCHOOL
OF MEDICINE; VISITING PHYSICIAN TO THE PHILADELPHIA GENERAL
HOSPITAL; VISITING PHYSICIAN FOR THE STUDY OF CARDIOVAS-
CULAR AFFECTIONS, PHILADELPHIA HOSPITAL FOR CONTAGIOUS
DISEASES; FORMERLY INSTRUCTOR IN MEDICINE, JEFFER-
SON MEDICAL COLLEGE; FORMERLY SPECIAL CARDIO-
VASCULAR EXAMINER, UNITED STATES ARMY.

ILLUSTRATED

MILITARY REFERENCES WITH THE PERMISSION OF
THE SURGEON GENERAL.



MADE IN U.S.A.

PHILADELPHIA
F. A. DAVIS COMPANY, PUBLISHERS

1921

COPYRIGHT, November, 1920

BY

F. A. DAVIS COMPANY

Copyright, Great Britain. All Rights Reserved

**PRESS OF
F. A. DAVIS COMPANY
PHILADELPHIA, U. S. A.**

YASSEL 33A

TO MY BROTHER
S. MACCUEEN SMITH, M.D.

ACKNOWLEDGMENTS

To Dr. Wm. L. Coplin, Director of the Jefferson Medical College Museum, for permission to photograph specimens;

To Dr. Allen J. Smith, of the University of Pennsylvania Medical School, for generous assistance in the selection of pathologic illustrations,

The writer expresses his indebtedness.

PREFACE

THIS is a treatise that deals with the heart and its affections—a book that does not presuppose a knowledge of the subject and that strives to encompass in small volume sufficient fundamentals of anatomy, physiology, pathology, diagnosis and treatment to give the busy physician a working knowledge of the more recent advances in studies of the heart.

Contact with medical men in civil life has taught me the need of a fundamental volume on the heart, as have also requests of medical students. Physicians engaged in military work at American training camps and abroad have voiced a similar sentiment.

Let that, then, be the *raison d'être* for this book, which is undertaken in the hope that it may fill a need. Perhaps a desire to be brief has caused me to pass rather hurriedly over chapters, the importance of which warrants more elaborate treatment. If this be so, let the brevity stimulate my readers to follow the studies and researches to be found in larger volumes.

S. C. S.

323 South Eighteenth Street,
Philadelphia, Pa.

80277

CONTENTS

CHAPTER I.

INTRODUCTION.

PAGE

The present day conception of heart affections—The place of auscultation in cardiac diagnosis—Instrumental methods of diagnosis—Their contribution to cardiology—Four preliminary postulates for students—Cardiac aphorism 1-3

CHAPTER II.

ANATOMY AND PHYSIOLOGY OF THE HEART.

Evolution of the heart—Size, weight and shape—Chambers of the heart—Valves of the heart—Circulation of the blood—The conduction system—Properties of heart muscle—The cardiac cycle—The cardiac nerves 4-20

CHAPTER III.

EXAMINATION OF THE PATIENT: GENERAL CONSIDERATIONS.

Extreme cardiac types—Diagnostic points—The presenting symptoms of heart affections—The attitude of the patient—The attitude of the physician—The keeping of records 21-33

CHAPTER IV.

EXAMINATION OF THE PATIENT (*Continued*): PREVIOUS HISTORY.

Acute infections of childhood—Infections of adolescence—Chronic systemic diseases—Septic absorption—Physical strain and emotional stress—Habits—"Inherited" heart disease 34-40

CHAPTER V.

EXAMINATION OF THE PATIENT (*Continued*): INSPECTION AND PALPATION.

Preparation of the patient—Inspection—Palpation 41-46

(vii)

CHAPTER VI.

EXAMINATION OF THE PATIENT (*Continued*): PALPATION
OF THE PULSE.

PAGE

Reason for feeling the pulse—Technique—Bimanual estimates—
Rate—Importance of rate-response to exercise—What con-
stitutes a natural rate-response—Rhythm—Volume 47-57

CHAPTER VII.

EXAMINATION OF THE PATIENT (*Continued*): CARDIAC
PERCUSSION AND MENSURATION.

Forms of percussion—Purpose of percussion—Malposition of
the heart—Mensuration 58-69

CHAPTER VIII.

EXAMINATION OF THE PATIENT (*Continued*): AUSCULTATION OF
THE HEART; MURMURS.

Purpose of auscultation—Technique—Stethoscopes—Natural heart
sounds—The puncta maxima—Cardiac murmurs—Tonal
Properties of Murmurs—Transmission of murmurs—Accen-
tuations—Reduplications—Friction rubs—Influence of exer-
cise on murmurs—Murmurs without significance—Febrile
murmurs—Murmurs of diagnostic import—Differentiation of
Murmurs, French Method—How to regard a murmur 70-81

CHAPTER IX.

LABORATORY AIDS IN DIAGNOSIS.

Urinalysis—Renal function test—Ophthalmoscopy—Serologic
reactions—Differential blood counts—Blood cultures—The
fluoroscope—The orthodiascope 82-88

CHAPTER X.

GRAPHIC METHODS OF EXAMINATION: THE POLYGRAM AND
ITS INTERPRETATION.

Advantages of the polygraph—The apparatus—Component parts
of the tracing—Technique—The normal arteriogram—The
normal phlebogram—Interpreting the polygram—The inter-
pretation of arteriograms—Dominant rhythm—Brief sug-
gestions in analysis—Abuse of the polygram 89-101

CHAPTER XI.

GRAPHIC METHODS OF EXAMINATION (*Continued*): THE ELECTRO-CARDIOGRAM AND ITS INTERPRETATION.

	PAGE
Definition—The principle—The apparatus—Questions the physician asks—Investigations that established electrocardiography—Clinical diagnoses confirmed—Diagnoses otherwise not possible—How to read the normal electrocardiogram—Interpreting the curves—Pathologic records—Brief suggestions in analyzing electrocardiograms—The use and abuse of electrocardiography	102-125

CHAPTER XII.

THE SIGNIFICANCE OF BLOOD-PRESSURE ESTIMATES.

Definition—Importance of comparative readings—The apparatus—Technique—Terms employed—Usual standards—Blood-pressure aids—Blood-pressure "Don'ts"—Treatment of hypertension	126-133
--	---------

CHAPTER XIII.

THE IRREGULAR PULSE.

General considerations—Sinus arrhythmia—Premature contractions—Paroxysmal tachycardia—Auricular flutter—Auricular fibrillation—Etiology—Prognosis—Treatment—Heart-block—Diagnosis—Prognosis—Treatment—Pulsus alternans—The general significance of pulse irregularities	134-163
---	---------

CHAPTER XIV.

PERICARDITIS.

The physiologic function of the pericardium—Classification of pericarditis—Etiology of acute pericarditis—Diagnosis of acute pericarditis—Pericarditis with effusion—Pericarditis with adhesions—Adherent pericardium—Treatment	164-188
---	---------

CHAPTER XV.

MYOCARDIAL AFFECTIONS.

Terms employed—Etiology—The induction of chronic myocardial change—Diagnosis of acute myocarditis—The recognition of chronic myocardial change—Treatment	189-214
--	---------

CONTENTS.

CHAPTER XVI.

ENDOCARDITIS.

PAGE

The defining of endocarditis—Morbidity anatomy—Etiology of acute endocarditis—Etiology of malignant endocarditis—Symptomatology—Physical signs—Diagnosis—Prognosis—Treatment 215-241

CHAPTER XVII.

CHRONIC VALVULAR DISEASE OF THE HEART.

General considerations—Etiology—Morbidity anatomy—Varieties of valvular disease—Incidence—Mitral insufficiency—Mitral stenosis—Aortic insufficiency—Aortic stenosis—Tricuspid valve lesions—Pulmonary valve lesions—Treatment 242-269

CHAPTER XVIII.

CONGENITAL HEART AFFECTIONS.

Varieties—Patulous foramen ovale—Perforate interventricular septum—Persistent ductus arteriosus—Valve defects—Dextrocardia—Congenital valvular disease—Conclusion 270-277

CHAPTER XIX.

ARTERIOSCLEROSIS.

Definition and terms—Circulatory effects—Etiology—Clinical recognition—Blood-pressure—Kidneys—Heart—Pulse—Eye—Prognosis—Treatment 278-306

CHAPTER XX.

ANEURISM.

Definition and varieties—Etiology—Acute aortitis: Symptoms—Physical signs—Diagnosis—Preaneurismal stage—Thoracic aneurism—General symptoms—Symptoms relative to site—Physical signs—Diagnosis—Prognosis—Treatment 307-336

CHAPTER XXI.

ANGINA PECTORIS.

The condition defined—Objectionable terms—Etiology—Morbidity anatomy—Symptoms—Physical signs—Diagnosis—Prognosis—Treatment 337-351

CONTENTS.

xi

CHAPTER XXII.

NEURO-CIRCULATORY ASTHENIA.

PAGE

The name—The syndrome in civil life—The syndrome in training camps—The syndrome in war—Predisposing conditions—Typical case record—Further points in analysis—Treatment—Conclusion	352-360
--	---------

CHAPTER XXIII.

"WHAT CAN BE DONE FOR HEART DISEASE?"

A false impression—The early recognition of heart disease—What cannot be done—What may be done—Prophylaxis—The patient's daily life—Correction—Conservation—Cardiac therapy—Rest—Exercise—Diet—Massage—Sanatorium treatment—Operation on septic foci—Balneotherapy—Drugs	361-392
--	---------

CHAPTER XXIV.

CARDIAC DRUGS.

Digitalis—Strophanthus—Epinephrin—The nitrites—Atropine—Morphine—Antisyphilitics—Iodides—Strychnine—Caffein—Alcohol—Camphorated oil—Anesthetics in heart affections—Chloroform—Ether—Nitrous oxide	393-411
--	---------

CHAPTER XXV.

CARDIAC CAMOUFLAGE.

Artificially induced disorders—The reason—The means employed—Simpler forms—Acetanilid—Atropine—Digitalis—Suspicious attitudes of mind—Genuine mental atmospheres	412-417
INDEX	419

ILLUSTRATIONS

FIG.	PAGE
The Heart (Frontispiece).	
1. Relation of Heart to Anterior Chest Wall	6
2. Right Auricle and Ventricle	8
3. Left Auricle and Ventricle	9
4. Lateral View of the Heart	10
5. The Valves of the Heart	13
6. The Excitation Wave	16
7. The Conduction System of the Heart	17
8. Form for Cardiovascular Record	31
9. Reverse Side of Cardiovascular Record	32
10. Rate Response to Exercise	52
11. Cardiac Displacement (<i>A</i>)	63
12. Cardiac Displacement (<i>B</i>)	64
13. Cardiac Displacement (<i>C</i>)	65
14. Cardiac Mensuration	67
15. The Ford Stethoscope	71
16. The Puncta Maxima	74
17. The Usual Outline of the Heart	84
18. Cardiac Enlargement	85
19. Effect of Respiration on the Heart	86
20. Groedel Orthodiascope	87
21. Mackenzie's Ink Polygraph	90
22. Polygram of an Apparently Normal Heart	90
23. The Jugular Bulb	91
24. Sinus Arrhythmia	98
25. Auricular Fibrillation	98
26. Dropped Beats	99
27. Complete Heart-block	99
28. Pulsus Alternans	100
29 The American Electrocardiographic Equipment	104
30. The Galvanometer and String-house	105
31. The Camera	107
32. The Three Leads	110
33. Component Parts of the Record	111
34. Normal Electrocardiogram	111
35. Sinus Arrhythmia	112
36. Right Ventricular Premature Contractions	113
37. Paroxysmal Tachycardia	113
38. Auricular Flutter	114
39. Auricular Fibrillation	115

FIG.	PAGE
40. Delayed Conduction	115
41. Dropped Beat	116
42. High-grade Heart-block	117
43. Arborization Block	118
44. From a Case of Mitral Stenosis	119
45. Auricular Enlargement	119
46. Left Ventricular Preponderance in a Case of Aortic Regurgitation	120
47. Vagus Nerve Compressed by Lymph Nodes	143
48. Hemopericardium	165
49. Acute (Villous) Pericarditis	169
50. Pericarditis with Effusion	173
51. The Cardiohepatic Angle	175
52. Probable Pericardial Effusion	177
53. Pericardial Adhesive Bands	181
54. Adherent Pericardium	183
55. Polyserositis—Pick's Disease	185
56. Cicatricial Myocarditis	195
57. Fibrous Myocarditis	202
58. Verrucose Valvulitis	218
59. Vegetative Mural and Valvular Endocarditis	219
60. Mitral Valve Vegetations	220
61. Aortic Valve Vegetations	221
62. Fibrous Fusion of Valves	222
63. Aortic Valve Leaflet Distorted by Vegetations	223
64. Fenestration of Aortic Leaflets	224
65. Perforation of an Aortic Leaflet	225
66. Apical Abscess	226
67. The Funnel-shaped Mitral	245
68. The Buttonhole Mitral	247
69. Fusion of Aortic Leaflets	255
70. Patulous Foramen Ovale	271
71. Imperfect Aortic Valve	275
72. Medial Calcification	284
73. Beginning Arterial Change	285
74. Arteriosclerotic Gangrene of Leg	287
75. The Arch of the Aorta	313
76. Aneurism of the Aorta	317
77. Aneurism	320
78. X-ray Photograph of Fig. 77	321
79. Aneurismal "Erosion"	326
80. Aneurismal "Erosion." Side view Fig. 79	327
81. Rupture of an Aneurism into the Esophagus	329
82. Foreign Body in the Heart	365
83. Influence of Digitalis on the Electrocardiogram	397

CHAPTER I.

Introduction.

HEART affections are being rewritten in that new nomenclature and broader understanding which has followed the investigations of the physicist, the physiologist, the pathologist and the anatomist, in their recent laboratory inquiries of the heart. The clinician has applied these investigations and advances at the bedside, and as a result many disorders of the cardiac mechanism, heretofore undiagnosed, can now be clinically recognized and classified for treatment.

The day has passed when heart disease and prognosis are determined by the detection of cardiac murmurs alone. The wisdom of an oracle is no longer believed to dwell in the mouth of a stethoscope. Auscultation of the precordium and the timing of adventitious sounds within the chest do not now hold first place in the determination of heart disease. Auscultation should hold sixth place in our methods of physical diagnosis, being preceded in importance by the logical order of investigation which is systematically employed in all modern medical examinations, *viz*: history-taking, inspection, palpation, percussion, mensuration. Then should auscultation be employed, to confirm observations made by these five methods and, perhaps, to adduce additional testimony of cardiac disease.

Supplementing the information thus elicited by the natural gifts of sight, touch and hearing, there

are now available for further investigation of the heart, if need be, other diagnostic methods. The *sphygmomanometer* records, in figures, changes in the blood-pressure of our patients—changes which are sometimes of pathologic significance. The *polygraph* records, in ink, the events transpiring in the right and in the left sides of the heart. The *electrocardiograph* records, in photograph, alterations that may occur in the course of the excitation wave which precedes the contraction of the various heart chambers. The *orthodiagraph* is an adaptation of the Röntgen rays which accurately determines any increase in the diameters of the heart or of the greater vessels.

However, these instruments of clinical precision are not to be considered absolutely necessary for the establishment of a clinically satisfactory diagnosis. In doubtful cases, or where there is a desire for further investigation, the newer diagnostic apparatus will of course be employed. The invaluable contribution which the electrocardiograph and the polygraph have made to science has been in the aid they afford in the *clinical* recognition of disturbances of the cardiac mechanism; they have classified such disturbances and have taught the physician to differentiate serious pulse irregularities from those which are quite harmless. Instrumental methods are not intended to entirely supplant the trained finger, the alert eye and the discriminating ear in the recognition of heart disease. Nor can their use supply to him who lacks it the finely-balanced quality of clinical *judgment*, which is the logical end result of carefully weighing the evidence obtained by history-

taking, inspection, palpation, percussion, mensuration and auscultation.

The points, then, to be impressed on the reader ere he peruses the successive chapters of this book are as follows:

1. Adopt a systematic method of heart examination, in which auscultation is to be the last, not the first, of the physical methods employed.

2. Attach little significance to the presence of systolic murmurs unless accompanied by other signs which indicate cardiac damage. One cannot estimate the degree of heart damage by adventitious sounds; nor is it wise, on the other hand, to assume that the heart is normal because it is free from murmurs.

3. It is the condition of the *all-essential heart muscle* and not the presence of an incidental heart sound, which determines the cardiac efficiency of an individual and which guides the physician in prognosis and treatment.

4. He who practices systematic routine in the examination of cardiac patients will arrive at a clinically-satisfactory diagnosis in the majority of instances, without the additional employment of modern instruments of clinical precision. And no matter what facilities may be at hand, he who lacks system in his examinations will have slipshod and uncertain results.

In conclusion, let us pursue our investigation of the heart and its affections by disabusing our minds of such earlier teachings as gave us the impression that a "murmur" is the indispensable factor in cardiac diagnosis, and in its place keep before us a sentence which might well be called today's

CARDIAC APHORISM:

*The muscle is of more importance than the murmur:
The rhythm is of more importance than the rate.*

CHAPTER II.

Anatomy and Physiology of the Heart.

“THE heart is a hollow muscular organ, situated anteriorly within the thoracic cavity, lying between the second and fifth interspaces. It extends eight centimeters to the left of the median line of the thorax and two centimeters to the right thereof. Its function is to propel the blood through the greater and lesser circulations of the body.”

In some such stereotyped phrase as this do texts on anatomy define the marvellous combination of muscle, nerve, membrane and chambers which evolved itself, embryonically, from a reduplication of curves in the primitive embryonic cardiac tube. If one thinks of the heart as thus derived, from that point in a blood-vessel where the deposit of an excess of nodal tissue gave rise to impulse; and if he continues to think of it as an architecturally-elaborate, highly-specialized evolution of an artery, he will better understand why pathologic conditions which affect the arteries so often affect the heart, and why maladies of the heart are not confined to that organ alone but may also involve the vessels of which it is a highly-elaborate structural part.

SIZE, WEIGHT AND SHAPE.

There are many circumstances which cause the heart to vary in size, in weight and in shape. Hence it is quite impossible to state that a heart, *normal for an individual*, shall have a specified weight, or that it shall conform to definite measurements, or that it

shall correspond to a given shape. *Sex* causes variations in the heart; it is larger in men than in women. *Stature* also causes variations. Short, stocky individuals have as a usual thing a broader heart than that which is found in the tall, thin person. In the latter the heart may be of a length which causes it to extend to the sixth interspace and be correspondingly narrow; to such an organ the term "dropped heart" has been applied. *Occupation*, in the demands which it may make upon the heart, has an influence on its size; the person who has never been accustomed to marked or continued physical exertion—the bookkeeper, for example—may be expected to have a heart which is smaller than that found in those accustomed to vigorous physical exercise. Those who are habitually engaged in arduous employments usually have efficient hearts; but if they suddenly abandon physical activity and lead a life of comparative ease, the heart may lose its quality of tone and seem to be increased in width. *Previous disease* often has an effect on the size of the organ. *General nutrition* if below par, may cause the heart to respond unnaturally to the demands thrown upon it, and the organ be larger than one would expect to find.

When one considers that a multiplicity of factors such as these may alter the size, weight and shape of a normally acting heart, one sees that allowance must be made for departures from the average figures. The total transverse diameter of the heart averages 12 centimeters.* The average weight is 10 ounces. The average capacity of the left ventricle is 4 ounces. The usual position of the heart is well within the tradi-

* $2\frac{1}{2}$ centimeters = 1 inch.

tional confines of the second and fifth interspaces (Fig. 1)—although it may extend quite beyond them in one direction or the other, as in “dropped heart,” and be in no wise considered pathologic.

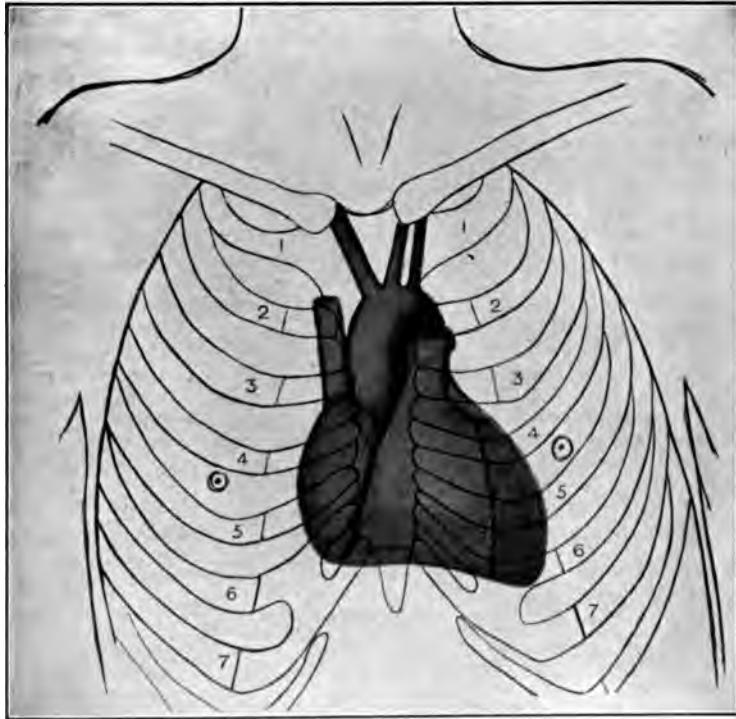


FIG. 1.—RELATION OF HEART TO ANTERIOR CHEST WALL.

The cardiac outlines were obtained by orthodiascopic projection. The right border of the heart is formed by the right auricle. (From Muckenzie.)

One must investigate the history, the habits and occupation of a patient—must employ inspection, palpation, percussion, exercise tests and the *sense of comparative values*—if one is to elicit definite information concerning each individual case. It is absurd

to attempt to appraise a heart simply by auscultation and, with a flourish of stethoscope, proclaim that the brawny day laborer has an "enlarged heart" or that the slender, slim young girl needs strenuous exercise to bring her heart to "normal" standards!

CHAMBERS OF THE HEART.

The interauricular and the interventricular septa which divide the organ in a longitudinal plane afford an anatomic basis for speaking of the "right heart" and the "left heart"—convenient phrases for memory, as we recall that the right heart is concerned with the venous blood of the lesser or pulmonary circulation, while the left heart receives and distributes the arterial blood of the greater or systemic circulation. It has been customary to speak of the upper chambers of either side as the "auricular reservoirs" (most elaborately designed, indeed, for mere "reservoir" purposes!); these communicate with the ventricles below through orifices called valves.

The distribution, direction and extent of the muscle fibers show many interesting modifications in the various chambers. The "internal architecture"—if one might thus refer to such structures as the columnae carnae, muscoli papillares and chordae tendineae, for example, which unite to form the operating mechanism of the mitral and tricuspid valves—is also subject to marked modification in the various chambers. Much of this can be learned at the dissecting table; little of it can be learned from printed words. Study of the illustrations (Figs. 2, 3 and 4) will refresh the memory on the relative size, thickness and structure of the various heart chambers.



FIG. 2.—RIGHT AURICLE AND VENTRICLE.

Both chambers laid open, the anterior wall of each having been removed. (From Chart of *Dr. G. H. Michael*.)

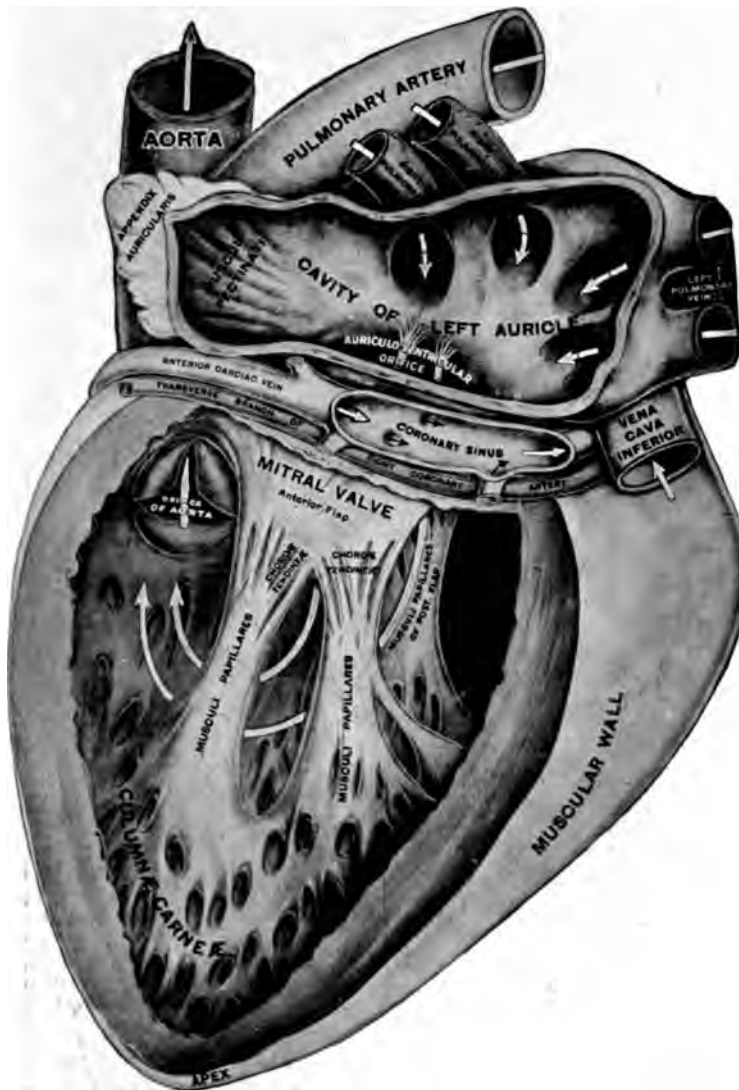


FIG. 3.—THE LEFT AURICLE AND VENTRICLE.
The arrows indicate the course of the blood. (From
Chart of *Dr. G. H. Michael.*)

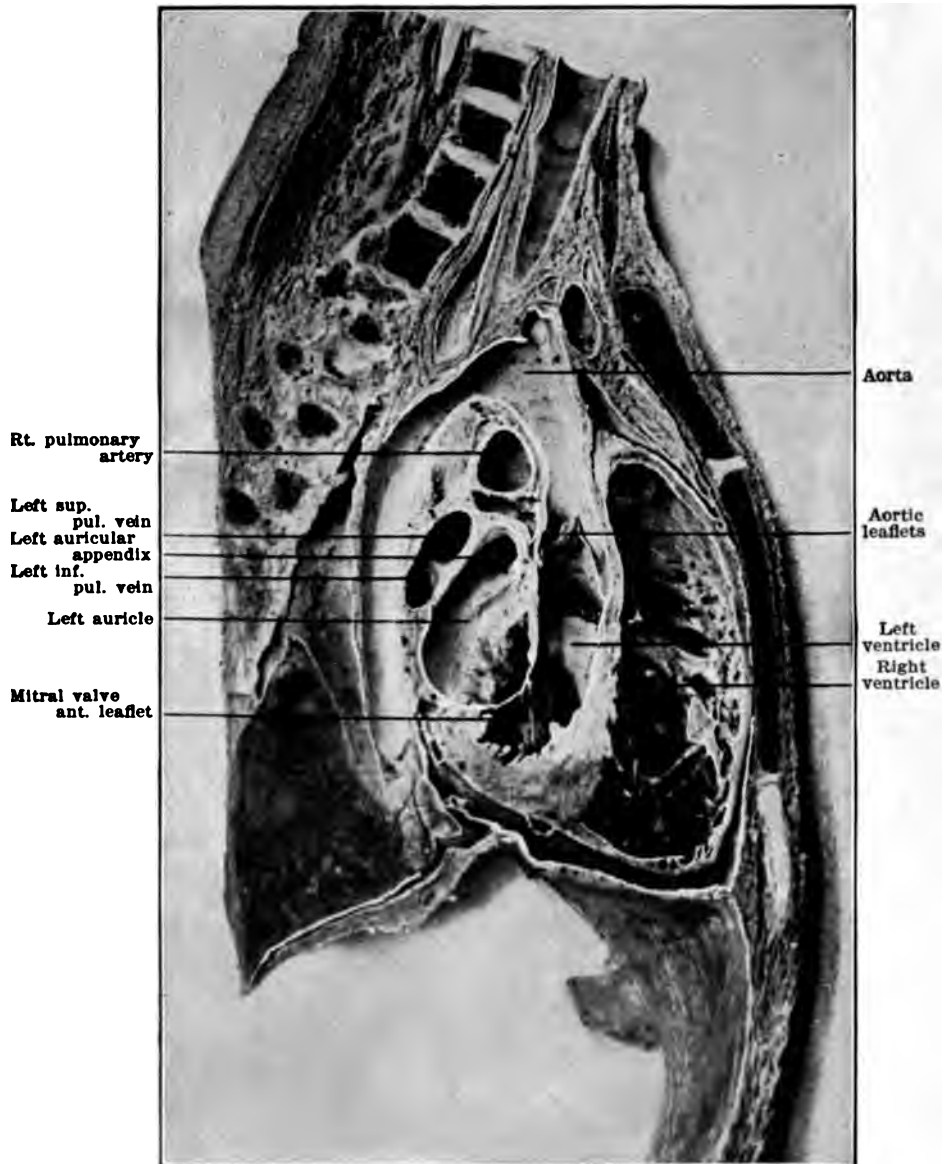


FIG. 4.—LATERAL VIEW OF THE HEART.

Obtained from section of a frozen specimen. Note position of the left auricle which is sometimes called "The posterior auricle." (From *Norris and Landis.*)

VALVES OF THE HEART.

That orifice situated in the right atrio-ventricular septum is closed by the tricuspid valve, which consists of three segments or "cusps" of triangular shape (Fig. 5). Its fellow of the opposite side is known as the mitral or bicuspid valve. It is larger and thicker than the tricuspid and consists of two segments.

The membranous leaflets of these valves are evolved from reduplications of the endocardium—the thin, translucent membrane which lines the cavities of the heart; they contain a few muscle fibers. The valves are reinforced by fibrous rings of comparative density where the leaflets join the heart muscle. It is interesting to note in passing that nature has paid particular attention to the reinforcement of the aortic valve of the ox, sheep and deer, to the extent of providing a crescent of bone around the anterior aortic cusp. The aortic or semilunar valve guards the orifice between the left ventricle and the aorta. It has three leaflets. The pulmonary valve also consists of three leaflets, and is located at the junction of the right ventricle with the pulmonary artery.

"Mitral disease" is a term freely used to describe an affection of this valve, which is of a size sufficient to usually admit the tip of three fingers. It is said that the mitral valve is damaged five times as often as are other valves of the heart, the averred reason for this being that it is subject to heavy "back-pressure" when the powerful and forcibly-acting left ventricle drives its content of blood into the circulation, thus tending to weaken the valve. The statement is quite at variance with physical laws in other

parts of the body, where the natural use of a structure develops and strengthens it. Nor can the statement be reconciled with investigations¹ which indicate that undamaged heart-structure is capable of putting forth 13 times the effort required in one day's life and yet emerge from the test apparently unaffected. It might be more nearly right to adopt the premise that mitral-valve damage is coincident with heart muscle affection—a local manifestation, in a valve, of a more widely spread condition—either from acute infections, absorption of toxins from chronic processes, or from long-sustained physical effort. For it is inconceivable that disease could invade the myocardium and fail to affect the overlying endocardium as well; and it is equally as improbable that infection would confine its ravages to the delicate valve-membrane alone and leave the contiguous myocardium uninvaded.

In speaking of the septum between the right and the left auricle the impression was perhaps conveyed that it is an imperforate dividing wall. Such is not always the case. The foramen ovale, which is patulous in the fetal circulation, may fail to close at birth and persist as an opening between the right and the left auricle. In the frankly patent cases, we see the phenomenon of "blue babies," a rare one of which may live to maturity and indeed to old age. Or, in other instances, the foramen ovale may close only imperfectly, remaining partly patulous through an apparently healthy life. Recent anatomic investigations indicate that this condition is found much more frequently than one would expect (see page 272). Under

¹ Levy: *Ztschr. f. klin. Med.*, 1896-97, xxxi, 320.

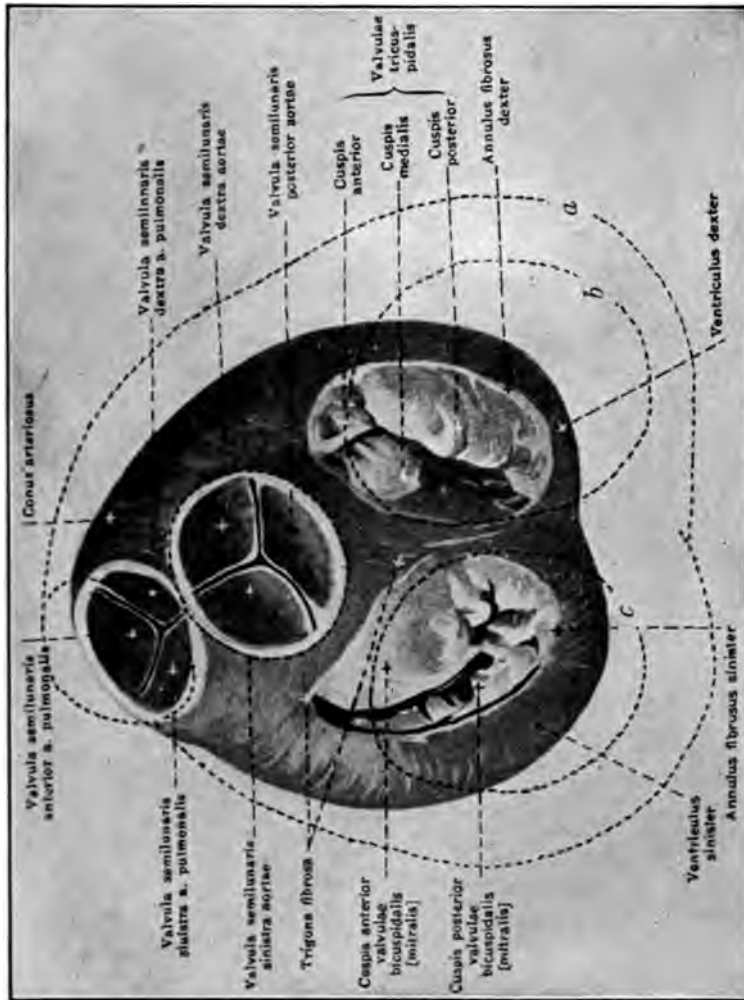


FIG. 5.—THE VALVES OF THE HEART

View from above, showing their relative size and position during systole. The dotted lines indicate the respective sizes during the rest period. (*Spalteholz.*)

physical strain or excessive emotion, or in a heart of relaxed tonicity, this imperfect closure may permit the admixture of venous with arterial blood, with attendant symptoms of cyanosis or dyspnea. Such instances may produce marked alterations in the usual heart sounds which are suggestive of murmurs—an observation which emphasizes the fatuity of building a diagnosis of heart disease solely upon the shifting sands of murmurs.

CIRCULATION OF THE BLOOD.

William Harvey, the London physiologist of the seventeenth century, published in 1628 his revolutionizing observation on the circulation of the blood. Arthur Keith, another London physiologist of the twentieth century, discovered in 1907 the sino-auricular node which initiates the contraction of the normal heart. One must review the old and new discoveries of these and other celebrated physiologists and anatomists, linking together successive revelations in their logical entirety, if one is to clearly understand the functioning heart.

Venous blood, be it remembered, is returned from the body to the right auricle by the ascending and descending venae cavae. It passes through the tricuspid valve into the right ventricle, through the pulmonary valve into the pulmonary artery, which carries the blood to the lungs for aëration. The oxygenated blood returns to the left auricle by way of the pulmonary veins. (The pulmonary "artery" carries venous blood and the pulmonary "vein" carries arterial blood—misnomers applied by the early anatomists). From the left auricle the fluid passes through

the mitral valve to the left ventricle, then through the aortic valve into the aorta and is thence distributed through the body.

The propulsion of the blood into the arteries is accomplished by simultaneous contraction of the ventricular chambers of the heart, the contraction being termed systole. The auricles of course also have a systole, and both auricles contract at the same time. It is a fraction of a second later—between 0.12 and 0.18 of a second—that both ventricles are in systole. Ventricular systole, in a heart which contracts at the rate of 75 beats per minute, occupies 0.3 of a second. Then follows a period of cardiac rest, known as diastole, which occupies 0.5 of a second (during the last one-tenth second of which the auricles contract). From this the rather interesting deduction can be made, inasmuch as the ratio of work to rest is as 3 to 5, that the heart is in contraction nine hours a day and in its rest period fifteen hours out of the twenty-four.

THE CONDUCTION SYSTEM.

But what mechanism regulates this rhythmical and synchronous contraction of the cardiac chambers? It is regulated by an excitation wave, which is transmitted to the chambers of the heart along a definite, established pathway, *the conduction system*. The impulse for contraction originates in the sino-auricular node, graphically named by the brilliant Lewis of London, the “pacemaker” of the heart. It is situated at the junction of the superior vena cava and the right auricle. From there the excitation wave spreads over the auricular wall in much the same

manner that concentric rings, from a pebble thrown in a brook, would spread to the banks on either side (see Fig. 6). Thus it reaches the *node of Tawara* (described in 1906 by the Japanese investigator whose name it bears), which is situated in the junctional tissues between the right auricle and ventricle (see Fig. 7). This node is the head of a neuro-muscular bundle of tissue of perhaps half an inch in length; from its location it is sometimes called the *atrio-*

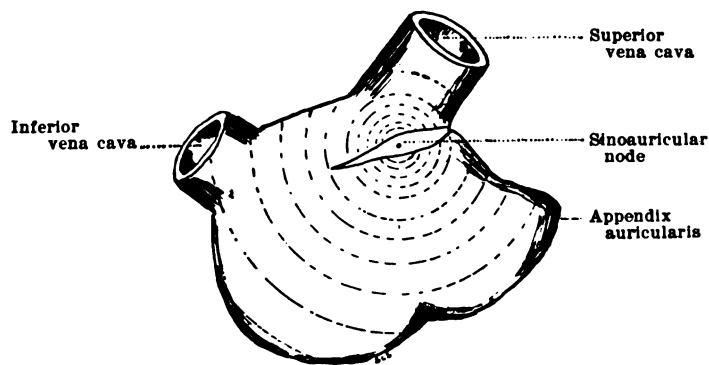


FIG. 6.—THE EXCITATION WAVE.

A diagram illustrating the spread of the excitation wave over the surface of the right auricle. The spread is almost uniform and follows the chief muscle bands. (After Lewis.)

ventricular bundle, although it is better known by the name of the anatomist who described it, *the bundle of His*. The bundle soon divides into right and left branches, the flat left branch piercing the interventricular septum to reach the left ventricle, while the round right branch conducts the stimulus for contraction to the right ventricle.

The right and left branches of the bundle terminate in fine arborizations known as the *fibers of Purkinje*, concerning which the anatomist of that

name wrote in 1845. Tawara showed us, in 1908, that these fibers are part of the conduction system and that they gradually fade into the ventricular muscle, where they discharge the impulse which now finally results in the contraction of the ventricles.

The stimulus for contraction varies in speed. It travels four times as fast in the ventricles, over neuromuscular tissue, as in the auricles; for in the latter situation it is conducted *via* muscular tissue alone.

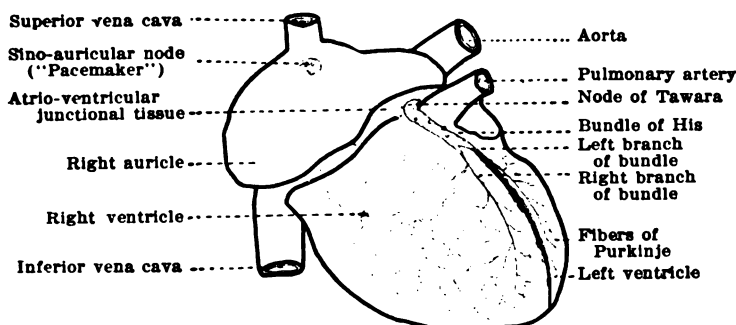


FIG. 7.—THE CONDUCTION SYSTEM OF THE HEART.

Showing in red the approximate relation of the more recently discovered structures to familiar anatomical divisions of the heart.

This is the pathway of conduction for a normal heart. It may be interfered with by disease. For example, it will be shown under *auricular fibrillation*, that in this condition the sino-auricular node is no longer the pacemaker of the heart and that the auricular rate is disordered by diseased foci situated in the muscular wall. There is another disturbance of conduction to be considered under its proper head, called *complete heart-block*, in which the bundle of His is affected and cannot conduct the impulse-for-contraction from auricle to ventricle. Under such a circum-

stance the unaffected portion of the bundle or even the ventricles themselves may initiate a rhythm of their own, and not depend on the pacemaker at all. In such an event the rates initiated by the lower part of the bundle are in the neighborhood of 45, while those originated by the ventricle are 30 or perhaps less.

THE PROPERTIES OF HEART MUSCLE.

We have seen in the foregoing paragraphs that heart muscle possesses the properties of: (1) *Stimulus production*; (2) *Conductivity*; (3) *Contractility*; (4) *Excitability*. To these we should add a fifth, the property of (5) *Tonicity*, by which heart muscle retains its tone and does not utterly relax during the rest period of the cardiac chambers. A loss in tonicity causes relaxation of muscle tissue; this in turn permits relaxation of the valve rings and prevents perfect closure of an orifice; thus there arise murmurs which are present when the patient is at ease, but which disappear on exercise. This is one of the phenomena produced by a loss in tonicity. These five properties are referred to collectively under the term of *the myogenic theory of heart muscle*. It is of clinical importance to bear in mind at this point that these five functions may be disturbed either singly or in combination, giving rise to the disorders discussed under, "The Irregular Pulse" (Chapter XIII).

THE CARDIAC CYCLE.

The *cardiac cycle* is that period which extends from the beginning of one contraction of the heart to the beginning of the next. It is well to commit to memory the paragraph which follows, for it is useful

when analyzing polygraphic tracings and electrocardiographic curves. It is valuable, too, to have in mind when studying patients at the bedside.

The cardiac cycle begins with the stimulus for contraction, which arises in the pacemaker and which is conducted along the auricular wall to the atrio-ventricular node and over the branches of the bundle of His. The mitral and the tricuspid valves now stand open, and the auricles contract. Next, the ventricles receive the stimulus from the fibers of Purkinje and contract, the contraction starting at the apex. Then the mitral and the tricuspid valves close. When the tension in the ventricles exceeds that in the aorta and in the pulmonary artery, the aortic and pulmonary valves open, and the *pulse period* begins. Ventricular systole is then completed. Comparative relaxation of the ventricles occurs, and when the pressure within them is lower than the pressure in the aorta and the pulmonary artery, the aortic and the pulmonary valves close and the pulse (sphygmic) period ends. The ventricles further relax, the mitral and the tricuspid valves open, and a new cycle begins.

THE CARDIAC NERVES.

The right and the left pneumogastric are the inhibitory nerves of the heart. It has been demonstrated recently that the right pneumogastric nerve sends the greater part of its fibers to the sino-auricular node. It is a distribution of much clinical significance; for inasmuch as the sino-auricular node governs the rate of the heart, pressure on the right pneumogastric nerve, which supplies the node, may cut short a paroxysm of tachycardia (page 141). The

left pneumogastric nerve sends a greater part of its fibers to the node of Tawara, thus particularly influencing conduction. The action of these inhibitory nerves of the heart is much referred to under the term "vagal influence."

The accelerator nerves of the heart are supplied by the sympathetic system, of which there are two plexuses. The superficial plexus lies beneath the arch of the aorta. The deep or great cardiac plexus lies behind the aortic arch, in front of the tracheal bifurcation.

The two coronary arteries, which arise from the aorta immediately above the aortic valve, nourish the heart (Fig. 68). They are about the size of a crow-quill. Partial occlusion, narrowing of the lumen, or arterial spasm of a coronary artery have been believed to be the exciting factors in angina pectoris.

CHAPTER III.

Examination of the Patient.

GENERAL CONSIDERATIONS.

EXTREME CARDIAC TYPES.

FROM the moment a patient enters the office, the alert physician may begin to make observations which will aid him in eventually determining the presence of heart affections.

For instance, in the case of an elderly person, he may observe that the step is measured and deliberate, as though in avoidance of rapid movements which, by disturbing the circulatory equilibrium, would cause giddiness and distress. The face may be either flushed or cyanosed, and bags hang under the lustreless eyes, as is frequently the case in patients with combined heart and kidney lesions. The respirations may be forced and shallow, and in sitting the patient may bend slightly forward, as though in studied protection of some malady within the chest. Pulsations may be visible in tortuous temporal arteries, and waves be present in the vessels of the neck.

Or as another example, take the young woman with the "neurotic" heart, who comes to consult the physician because of fainting attacks, faraway sensations, etc. She will present none of the signs by which the elderly cardiopath may be recognized. Her carriage will likely be erect, and her color natural. But the observant physician may notice that her move-

ments are quick, as though in response to a highly emotional state; her respirations hurried; her thyroid gland quite apparent and fine tremors present in her finger tips—all suggestive of the tachycardia which is associated with thyrotoxicosis.

Thus by careful observation of gait, bearing, attitude and superficial physical signs, one may classify the malady which brings the patient to the consulting office, and be on the alert for more definite symptoms which may be elicited as the examination proceeds.

DIAGNOSTIC POINTS.

One may observe other points while the patient relates his symptoms—points quite suggestive of definite heart lesions. The head may nod with each pulsation of the heart in aortic insufficiency; this affection, too, gives more pallor than does any other valvular disease of the heart. Radial arteries that are prominent or of a tortuous course will suggest arteriosclerosis, as they move laterally in the direction of the ulna with each impulse. A husky voice should cause one to think of the distended auricle of auricular fibrillation or of an aneurism, either of which sometimes press upon the recurrent laryngeal nerve, producing this symptom. Should four or five waves be counted in the jugular vein to one in a slowly-pulsating carotid, it will be interesting to determine whether this be a sign of heart-block. A brassy cough, with evidence of congestion, should remind the physician of a mitral lesion. Thyroid enlargement and protruding eyes at once suggest hyperthyroidism, with its attendant rapid heart. The widened space between the eyelids, the prominent cheek bones, the

transparent skin and the hectic flush of tuberculosis should lead the physician to anticipate the overacting heart of this condition. Should attention be arrested by characteristic scars or copper colored spots, search should be made for evidence that will convict syphilis of adding still another victim to its growing list of heart wrecks.

PRESENTING SYMPTOMS OF HEART AFFECTIONS.

The foregoing observations are simply diagnostic straws, which serve perhaps no other purpose than that of adding to the interest in a case. The best indication, before physical examination, that the patient actually suffers from a heart malady will be found in the *presenting symptoms*—*those symptoms of which the patient actively complains.*

As indicative of the order of frequency and the relative importance of the *earlier* presenting symptoms of heart affections, the following table is presented:

INCIDENCE OF PRESENTING SYMPTOMS IN 500 CONSECUTIVE REJECTIONS FROM MILITARY SERVICE.		Per cent.
Precordial pain	68.2	
Giddiness	66.6	
Palpitation	66.4	
Cough	34.4	
Dyspnea	31.4	
Fainting	29.5	
Edema	7.8	

These figures I gathered from a group of young men but recently drafted from civil life, who had physical breakdowns of varying degree during their first few weeks of training at a military camp. It is significant that precordial pain, giddiness and

palpitation were present in two out of three, of these recruits; cough, dyspnea and fainting were symptoms in one out of three. Only one out of every fourteen had edema, for the reason that edema is one of the later symptoms of heart affections, and these were early cases. We may now briefly discuss the above and other presenting symptoms.

(1) *Inability to perform customary tasks without distress* was the dominant symptom in this group of men. It was the reason for the vast majority of them being referred to the cardiovascular board—and hence it is *above all the most important presenting symptom*. The men “broke” under drills, marches and double time: they were unable to withstand a degree of physical effort which imposed no hardship on hundreds of others. The routine of military life uncovered heart affections which were not revealed when the man was first recruited from civil life. After the first cardiac break, a few were unable to perform even the simple act of climbing stairs or bending to lace shoes, without symptoms of distress.

(2) *Precordial oppression and pain* were present in 76 per cent. of the men with mitral lesions: in 72 per cent of those with cardiac enlargement (the cause of which was not always demonstrable); 52 per cent. of early aortic lesions complained of pain. Hence precordial oppression or pain is an early and important symptom of heart affections, and should never be lightly passed over without a thorough search for its cause. It may be due to pulmonary conditions or to pleural irritations; still less frequently it is caused by fatigue of the breast muscles; rarely indeed can the diagnosis of “intercostal neuralgia” and “gastric

flatulence" be made an excuse for neglect in searching for the probable cardiac origin of precordial pain.

Precordial hyperesthesia—tenderness of the pectoral muscles—might be mentioned in this connection. It is a sign that is not unusual in cardiac conditions, and is elicited by grasping the upper border of the pectoralis major muscle. It is often found in cardiac neuroses.

(3) *Giddiness*—vertigo or dizziness—was present in 71.5 per cent. of the men with mitral lesions; in 66.9 per cent. of those with cardiac enlargement and in 58.8 per cent. of early aortic lesions. Any of these affections will interfere with circulatory efficiency and giddiness arise as a result of sudden change of posture, or muscular exertion. This disturbance of the sense of stability also occurs in Meniere's disease, —a rare affection—and as a result of gastric, ocular or nerve disorders; but in the latter conditions it is not as constant a symptom as it is in cardiac affections.

(4) *Palpitation*—by which I mean a periodic rapidity of heart-rate of which the patient is uncomfortably conscious—was present in 67.5 per cent. of mitral lesions; in 69.1 per cent. of cases of cardiac enlargement and in 47 per cent. of the aortic lesions in the series. To be of significance, periods of palpitation should arise in the absence of muscular exertion and emotion. It is of passing interest to note that Darwin, in speaking of the influence of fear in his essay on "The Origin of the Emotions," refers to palpitation as follows: "the heart beats quickly and violently, so that it *palpitates or knocks against the ribs*; but it is doubtful if it then works more

efficiently than usual, for the skin becomes pale as during incipient faintness."

(5) *Cough* arises as the result of so many conditions that one must rule out of consideration the more common causes before attributing any cardiac significance to cough. Pharyngitis, laryngitis, bronchitis, pleuritis and other acute and chronic pulmonary conditions are the most frequent factors. Unusual causes are a relaxed uvula which irritates the pharyngeal wall, and impacted wax in the external ear which, by stimulation of the auditory branch of the pneumogastric nerve, produces cough. Caseous material in the follicles of tonsils not infrequently causes cough. The smoking of tobacco and the habit of mouth breathing obviously induce cough. When such apparent causes are ruled out of consideration, *cough is probably an evidence of heart affections* and is frequently associated with râles at the base of the lungs posteriorly—an early and valuable sign of beginning circulatory failure.

Of the mitral lesions found among the 500 cases which form the bases of this discussion, 35.7 per cent. had cough not due to obvious cause; 38.3 per cent. of the cases of cardiac enlargement presented the symptom, as did also 23.5 per cent. of the aortic lesions.

(6) *Dyspnea*, as an early symptom of heart affections, is usually transitory and is precipitated by some trivial exertion; this differentiates it from the labored breathing of bronchitis, pulmonary edema, toxemia and acute lung infections in which effort is not required to produce the symptom. An enlarged liver, ascites or other intra-abdominal enlargements may

cause dyspnea. In the absence of such demonstrable causes, dyspnea was present in 37.7 per cent. of the mitral lesions, in 26.3 per cent. of the cases of cardiac enlargement and in 35.2 per cent. of the aortic lesions in this series.

"*Cardiac sleep-start*" might here be mentioned, as it is caused by a paroxysm of dyspnea. It cannot be catalogued as an early symptom, however. The patient is suddenly awakened from sound sleep and, with a start, sits upright in bed. The period of apnea terminates after a short interval. Such experiences often cause the patient to elect to sleep in the sitting posture, rather than the recumbent. Cardiac sleep-start should not be confused with the sense of heart oppression which many people experience when they turn on their left side during sleep. This oppression is often accompanied by dreams of falling through space—and the sleeper awakens suddenly to change position, or may forcibly throw himself on the other side without fully awakening. But here the urgent absence of breath, which characterizes the genuine cardiac sleep-start, is lacking.

(7) *Fainting*, as a presenting symptom, in the preceding table (page 23) shows an interesting discrepancy between a mitral lesion incidence of 27.1 per cent. and an aortic incidence of 5.8 per cent. Fainting is a frequent symptom in the emotionally high-strung, in the constitutionally inferior and in persons who suffer from gastro-intestinal conditions. The fainting which is due to early circulatory disturbance is not to be confused with the periods of unconsciousness which arise, often without apparent cause, in far-advanced heart affections, in which unconsciousness is the re-

sult of coincident damage to the bundle of His and to the cerebral vessels.

(8) *Edema* of the feet or ankles, which appears at the close of the day and which has a tendency to disappear when the patient is at rest in bed, is characteristic of moderately-advanced and advanced heart affections. Scrotal edema, ascites and anasarca are deepening degrees of extravasation of fluid in tissues.

(9) *Cyanosis* is not an early symptom of heart affections. Excluding neurocirculatory asthenia, in which moderate cyanosis is a part of the symptom-complex, cyanosis usually occurs in association with other well-marked physical signs which leave no doubt as to the cardiovascular cause of the malady. One must of course bear in mind that cyanosis is not always of cardiac significance, and that it may occur in pulmonary affections; as a result of intracranial conditions; as a manifestation of toxic absorption; and also in the primitive emotion of rage. Cyanosis may be extreme in congenital malformations of the heart and in lesions of the pulmonary and tricuspid valves. An increase in the number of red cells—polycythemia—is an occasional cause of a puzzling and symptom-free cyanosis.

(10) "*Dropped Beats*" (actual dropped beats are rare), or a sensation of the "heart turning over," or of its "suddenly stopping," are very frequent words by which patients seek to express the phenomena of premature contractions. A premature contraction is one of many forms of pulse irregularities (see Chapter XIII). Every pulse irregularity should be thoroughly studied in order to determine its nature and to determine whether it is progressive. The electro-

cardiograph or polygraph may be required to elucidate confusing pulse irregularities.

(11) *Cheyne-Stokes breathing*, as a symptom of cardiac affections, occurs in advanced cases. It is a late—often a terminal—symptom in patients suffering from heart damage. In this condition periods of paroxysmal dyspnea alternate with apnea. There is a respiratory cycle—at first the breaths are faint, shallow and infrequent; gradually they increase in intensity and frequency, then as gradually decline to a period of respiratory silence which may last from 5 to 40 seconds, after which the cycle is repeated. Cheyne-Stokes respiration, in cardiac conditions, occurs usually at night, but may be continuous. It is also seen in coma which arises from affections of the nerve centers, uremia, narcotic poisons, acute infections, etc.

(12) *Other presenting symptoms* of cardiac significance referable to the brain or to the nervous, gastro-intestinal and genito-urinary systems, are discussed elsewhere in these pages under the affection to which they more especially apply.

THE ATTITUDE OF THE PATIENT.

The patient goes to the physician for the purpose of learning what bearing his presenting symptoms have upon his future. He wishes to know whether or not they mean heart disease—a term of terror to the layman, in whose mind it conjures the picture of sudden death while in the midst of the pleasures or vocations of life. Perhaps he has been alarmed by the illness of a friend with similar symptoms, or he may have been thrown into a panic by life-insurance

rejection. Whether his frame of mind reveals it or not, he stands as a prisoner at the bar, awaiting verdict. What should be the attitude of his counsel, judge and jury—the physician?

THE ATTITUDE OF THE PHYSICIAN.

The physician's attitude should be one of quiet reassurance. One cannot hope to succeed in heart work if one has an abrupt manner, disinterested attitude, overbearing voice or that unfortunate air of diagnostic finality. He who would get the most information out of a heart examination must take pains that his manner in no way alters the rate, rhythm or volume of the patient's heart. The cardiovascular examiner should be a physician whose quiet bearing and genuine interest inspire confidence, trust and hope. To avoid alarming his patient he should never employ the enigmatic shake of the head; he should not prolong auscultation; he should regard his patient with human interest and not as a pathologic specimen; and he should never use the alarming term "heart disease" in discussing a patient's condition with the patient.

THE KEEPING OF RECORDS.

It is desirable to keep a case-record of cardiac examinations. A record is necessary for future reference in further consultations and also for the purpose of definitely noting progress. It may save humiliation in medico-legal cases. It teaches system and encourages order. The record may be kept in an ordinary blank book, or a card-index system may be used.

THIS SPACE FOR POLYGRAPHIC TRACINGS
OR ELECTRO-CARDIOGRAPHIC
CURVES

Date Graphic Record by

NAME No.
CAMP DATE

Cardiovascular Examination

Original Record for Military Files of

DR. S. CALVIN SMITH,

PHILADELPHIA

Rank Co Regiment

Home Address

Age Race Nativity

Height Usual Civil
Weight Occupation

Date Entered Service { Enlisted.
Drafted

Referred by { Form 88
S. C. D.
Hospital Case

Referred for

DIAGNOSIS:—

Authority: Manual Page Par

RECOMMENDATION:—

In Line of Duty?

Recruiting Officer Blamable?

HOSPITAL CASE:—

Ward Bed

Entered Hospital

Admission Diagnosis

Decision of S. C. D. Board:

Pending Receipt of S. C. { Return to Duty
D. Papers { Remain in Qts. ...
Retain in Ward ...

Recording Clerk

Reviewed and Approved

by
Special Cardiovascular Examiner.

FOLD

FIG. 8.—FORM FOR CARDIOVASCULAR RECORD.

The form is folded in the center for filing, after being filled
out on both sides.

PREVIOUS HISTORY. (Put Down Age at Which Illness Occurred)

- | | | |
|-------------------------|--------------------------|--|
| 1. Abscessed Ears..... | 8. Pertussis | HABITS { Tobacco..... Alcohol.....
Drugs.....Sleep..... |
| 2. Abscessed Teeth..... | 9. Pneumonia | |
| 3. Chorea | 10. Rheumatic Fever..... | Complains of.....Duration |
| 4. Diphtheria | 11. Scarletina..... | Stopped Work.....Took to Bed..... |
| 5. Gonorrhea | 12. Syphilis | Palpitation.....Pain.....Dyspnea.....Cough..... |
| 6. Measles..... | 13. Tonsillitis..... | Fainting.....Giddiness.....Flushing.....Edema..... |
| 7. Mumps..... | 14. Typhoid Fever..... | Withstand Drills?.....Double Time?.....Marches?..... |

General Appearance {	Healthy, Pallid. Florid, Cyanotic.	Development {	Muscular, Robust. Slender, Frail.	Expression {	Intelligent, Alert. Apathetic, Anxious.
-----------------------------	---------------------------------------	----------------------	--------------------------------------	---------------------	--

Pulse { Regular } Volume..... Visible
 { Irregular } Pulsations

Arteries {	Relaxed. Infiltrated. Elastic. Indurated.	PRECORDIAL IMPULSE {	Circumscribed {	Normal Impact Thrusting Faint	Diffuse {	Heaving Impact Thrusting Slapping Faint
-------------------	--	-----------------------------	-----------------	-------------------------------------	------------------	--

		Faint		
		Brachial	Femoral	
Arrhythmia	None			
	Disappears on Exercise			
	Increases on Exercise			
	Following Return from Exercise			
		Blood Pressure	Systolic.....	
			Diastolic.....	
			Pulse Pressure.....	

Thrills { Basal. Systolic { Apical. Presystolic	M
---	---

Maximum Cardiac Impulse.....int.	Cardiac Borders	(2).....	Transverse Diameter of Arch
.....C. M. to L. of M. S. Line.		(4).....	B. E..... A. E.....(5)
Cardio-Hepatic Angle.....			A

Murmurs	{ Apical Basal	Systolic.	Presystolic.	Transmission	{ Over Precordia: To Axilla: To Back: Along Heart Border: To Vessels of Neck
		Diastolic.	Systolic.....		

Accentuations:—A.....; T.....; P.....; M.....

Notes	Standing	Recumbent	Immediately After Exercise	Dyspnea Immediately After Exercise	Five Second Rate Return						Two Minutes Later (Recumbent)
Respiratory				None	10	15	20	25	30	45	
Ventricular				Moderate							
Radial				Urgent							
Deficit ?				Lastedmin.							

CLINICAL TESTS:—

Wassermann **Urinalysis**

X-Ray **Renal Function**

Graphic Record

REMARKS:

DIAGNOSIS:

.....

.....

FIG. 9.—REVERSE SIDE OF CARDIOVASCULAR RECORD.

The loose-leaf system, cross-indexed for "patients" and "diseases," and further indexed for "open accounts" and "closed accounts" is perhaps the most convenient of all. When it is desired to standardize examinations for the purpose of gathering statistics for the compilation of clinical data, it is useful to use a blank form. When doing much cardiovascular work the blank form saves time, as on it the secretary can record the findings of the physician with a few strokes of the pen when the observations are called off by the examiner. Military necessity caused the writer to devise such a blank (Figs. 8 and 9) which proved satisfactory in thousands of army examinations. With a few changes and the addition of space in which to outline treatment, progress, etc., it can readily be adapted to a loose-leaf system for private and hospital work.

CHAPTER IV.

Examination of the Patient (*Continued*).

PREVIOUS HISTORY.

ACUTE INFECTIONS OF CHILDHOOD.

THE term *heart disease*, as usually employed implies a chronic affection of either the valves or muscle of the organ. Being chronic, by the time it presents at the physician's office, it is the more or less remote result of antecedent damage. Hence it is important to know the previous history of a patient and to supplement this information with a knowledge of the manner in which previous diseases have been found to commonly affect the heart. Cardiac damage may have had its inception in the acute diseases of childhood, such as scarlet fever, chorea, diphtheria, measles, etc., or it may have arisen during acute infectious processes of adolescence. The heart, wearied by the demands thrown upon it during the progress of these diseases, and perhaps infected itself at the same time, had two other circumstances with which to contend—the first being an insufficient period of convalescence in which to recover itself; the second, the labor imposed on a weakened organ by the systemic demands of the growing child and by the unrestrained activities of childhood. Consequently, such a heart comes to adult life more or less impaired. In analyzing my military records I have concluded

that the remote infections of childhood have been responsible for heart symptoms sufficient to reject the applicant from active military service in 1.5 per cent. of the total number examined, or about 50 per cent. of the total number rejected. The infections of childhood are further discussed on page 190.

INFECTIONS OF ADOLESCENCE.

Among what might be called infections of adolescence, rheumatic fever looms large as a provocative cause of heart damage. It has been estimated that 20 per cent. of those who suffer from rheumatic fever develop heart affections. Rheumatic fever is not to be confounded with the hybrid term "rheumatism," which is often a cloak that covers chronic septic absorption, with its attendant muscular aches and pains, from some focus within the body. The physician will do well, when he encounters the term "rheumatism" in previous history, to ascertain the number of weeks the patient was in bed with the affliction and inquire as to fever, condition of joints, etc., at that time in order to determine the history of acute rheumatic fever. Tonsillitis, the frequent precursor of rheumatic fever, is also provocative of heart lesions. Influenza is an increasingly common cause of cardiac damage. It has been stated that pneumonia induces auricular fibrillation in 10 per cent. of its victims. Typhoid fever frequently involves the myocardium, as also does chorea. The incidence of gonorrheal endocarditis is sufficient to warrant heart care during the progress of the more virulent instances of specific urethritis.

CHRONIC SYSTEMIC DISEASES.

Syphilis directs much of its attack against the cardiovascular system, as is witnessed by the frequency of its history in aneurism and heart-block and, less often, in arteriosclerosis and angina pectoris. Considerable time elapses between the initial lesion and the detection of cardiovascular syphilis, believed by many clinicians to be perhaps fifteen years. Exophthalmic goiter affects the heart, it is held, through the toxicity of the thyroid hypersecretion. The wasting disease tuberculosis is incessant in its demands on heart muscle and thus induces cardiac enlargement, often associated with valve-leakage, in the effort which the heart makes to supply the starving bodily tissues. Tuberculous patients may occasionally present what is apparently a displacement of the heart toward the affected lung; this is due to retractive changes in lung tissue. The toxins of gout may be reflected in cardiac disturbances. The association between nephritis and cardiac affections is shown by the classical hyphenated term "cardiovascular-renal disease." Mineral poisons, such as arsenic and lead, frequently induce permanent cardiac damage, the latter quite often delaying conduction.

SEPTIC ABSORPTION.

Foci of suppuration should be sought for in all obscure derangements of the heart. Chronically diseased tonsils may harbor the focus. Much more frequently than is supposed, the trouble lies in an unsuspected abscess at the apex of a symptom-free tooth or teeth. Faulty dentistry is an especially unfortunate

cause, inasmuch as recent dental work may give a patient a sense of security in work improperly done. Crowned teeth should be regarded with suspicion and examined by the *x*-ray. Even in the absence of definite abscess formation, one should bear in mind that the peridental membrane may harbor the *Streptococcus hemolyticus* or *Streptococcus viridans*—and not exclude from suspicion teeth which are devitalized or “pulpless.” Pyorrhea furnishes a focus for absorption that may be reflected in the heart. So may abscesses and chronic joint diseases. Cardiac disturbances are repeatedly seen to re-adjust themselves following operative procedures on a discharging ear, a septic gall-bladder or chronically inflamed appendix.

PHYSICAL STRAIN AND EMOTIONAL STRESS.

Continued *physical strain*, in excess of effort which a given heart has been taught to endure, is undoubtedly a cause of heart maladies. The recent war is replete with instances, both at home and abroad, of men of sedentary habits who were suddenly whirled into lives of military activity, with the result that they had cardiac breaks under the strain of the new and unaccustomed life (see Chap. XXII). Nor should there be any question of the part taken by *emotional stress* in the production of disordered action of the heart—such stress as exists, for example, in domestic calamities, shocking news, profound anxiety or distressing loneliness. Whether these maladies be designated by the newer term of “effort syndrome” or “neuro-circulatory asthenia,” or whether they be included under the older phrases of “neurotic heart”

or "adolescent heart," the provocative factors remain the same, *viz*: physical strain and emotional stress.

HABITS.

Certain habits have a pernicious action on the heart; others, through the medical usage of years, have a blame attached to them which investigation scarcely sustains.

Alcohol primarily increases the heart-rate and later induces a loss in the force of the ventricular contraction. Long continued use makes it a contributory cause of arteriosclerosis.

Tobacco aggravates premature contractions in hearts already affected from other causes; it also increases the pulse-rate in already affected hearts. In those unaccustomed to its use it raises pulse-rate, until systemic tolerance is established. There is no proof yet adduced that it adversely affects a normal heart unless used immoderately.

I enquired concerning tobacco smoking among one thousand recruits, recently drafted to one of the military camps from all conditions of civil life, and elicited the following information. Eighty-seven per cent. of them smoked, on an average of 11 times a day. The average pulse rate of abstainers was 82 per minute; of tobacco users, 85 per minute. The incidence of heart affections was found to be considerably higher among abstainers. This paradox, however, is explained by the fact that many abstainers had stopped using tobacco when it seemed to affect their hearts unfavorably, causing pulse irregularity or periods of rapid heart action.

The hearts of heavy smokers have been examined after death, and the only finding which was at all constant and otherwise unaccounted for was a shortening of the papillary muscles—a change the degree of which is certainly difficult to estimate after death. Until more definite evidence is forthcoming, a safe rule would be to interdict tobacco if its use produces unpleasant sensations or manifestations of nerve disorder in a patient; and it is equally safe to adopt the premise that one should not arbitrarily interdict the accustomed habit of years unless there be evidence that it provokes additional symptoms or signs in that particular patient. One should be able to determine this by a temporary abandonment of the habit for a fortnight.

Excessive physical exercise might here be mentioned as a pernicious habit, especially in these days of the too strenuous life. If exercise be judiciously indulged in, it works benefit and health to the majority of people; but to force oneself, when wearied or indisposed, into strenuous games is certainly ill advised. Athletic contests, in which physical effort must often be continued far beyond the point of fatigue and exhaustion, are responsible for the occurrence of “athlete’s heart.” The heart of an athlete is one in which sustained physical effort has so increased the demands on heart muscle that the muscle property of tonicity is impaired. In consequence of this myocardial relaxation, the heart is incapable of meeting even the demands of moderate exercise. Systolic murmurs are often heard at the apex of the heart, and pulse irregularities may arise. The over-strained cardiac tissue is often an easy prey for infections which manifest themselves in actual heart damage.

Habit-forming drugs which are extensively used include analgesics, the cardiac-depressant coal tar derivatives such as acetanilid (see Chapter XXV), phenacetin, antipyrin, etc., and opium preparations, which also act in slowing heart-rate. Cocaine and caffeine are stimulants, increasing the pulse-rate.

"INHERITED" HEART DISEASE.

It is rather the rule for patients to state that "heart disease runs in their family" and conclude from this premise that they have inherited a cardiac malady. Their minds should be disabused of the idea. With the debatable exception of congenital syphilis, heart affections are not inherited. Valvular disease of the right heart, which may sometimes exist at birth, may on first thought seem to be an exception to this statement: but it is a congenital result of fetal endocarditis and does not at all imply the existence of the same malady on the part of the mother. Certainly, one may inherit the family characteristic of inefficient heart muscle, just as one may inherit slender and delicate muscular structures in other parts of the body; one may exhibit the heritage of weak cardiac nerves, just as one may present the family *tic*. One may go through life with this cardiac embarrassment, or may reconstruct one's inheritance and make it into cardiac sufficiency. But in its final analysis heart disease is not inherited, it is acquired.

CHAPTER V.

Examination of the Patient (*Continued*).

INSPECTION AND PALPATION.

PREPARATION OF THE PATIENT.

It is folly to attempt a cardiac examination when the patient has his clothes on. It not only utterly deprives the physician of the valuable information to be gained from inspection, but it also interferes with the clearness of the percussion-note and with the appreciation of resistance to attempt percussion through intervening layers of clothing. If the physician has neither time nor inclination to properly prepare the patient for examination, it is better that he content himself with obtaining what information the pulse affords and postpone the complete cardiac examination until time and opportunity permit him to be just to the patient and fair to himself.

All clothing should be removed to the waist line. Considerations of delicacy will suggest that patients of the gentler sex be permitted to wear a dressing gown or an examining cape, which can be drawn to the side as examination demands.

The patient should stand before the physician with direct light falling upon his chest. He should stand at ease, with arms at side, and not attempt assistance in the examination by twisting to one side or the other, for he thus distorts anatomic landmarks and

increases the muscular resistance of the chest. If unable to stand, the patient should sit relaxed, on a straight, armless chair; if he sits on the side of a bed he should not be permitted to support himself with either arm, for the reasons just stated.

It is quite necessary for the physician to get his first impressions while the patient is in the erect or sitting posture, if at all possible; it is an essential part of systematic, routine examination. Observations thus made will of course be supplemented later on with the patient recumbent. For it is to be remembered that the heart is subject to postural shift, and that, therefore, information obtained solely when the patient is in the reclining position is unreliable and may lead to erroneous conclusions.

INSPECTION.

In gathering cardiac evidence from inspection it is well to proceed from above downward. The *head* may nod with each pulsation of the heart. The patient's *general appearance* may be pallid or cyanosed. The *eyes* may show the arcus senilis, or there may be sluggish or irregular pupils suggesting a systemic infection which would have an influence upon the heart. The *mouth* frequently gives evidence of pyorrhea, and the condition of the teeth may excite suspicion. The appearance of the mucous membrane and of the tonsils should next be noted. There may be *visible pulsations of the vessels of the neck*. If the carotids be forcible and quickly rising they suggest an aortic lesion; should the waves vary in volume or if there be an apparent deficit between the carotid and ventricular pulsations, mental note should be made

that auricular fibrillation is a possibility in the case.

The *shape of the chest* should now be noted in regard to any change that malformations might make in the normal relations of the heart. Should the *thyroid gland* be even slightly enlarged, look for widening of the palpebral orifice, for a tremor in the protruded tongue and for a tremor in the finger-tips of the outstretched hands. In addition, seek for von Graefe's sign of exophthalmic goiter, in which the eyes, directed towards the ceiling, will follow the finger more rapidly in a downward course than will the upper lid. The *supra-sternal notch* may pulsate, as may also the 2d interspaces to the right and to the left of the sternum, when viewed laterally—evidence of aortic dilatation or of aneurism of the arch; (Lateral inspection of this area is also to be performed when the patient is later recumbent, as the information may then be more clearly elicited).

The precordial impulse should next be noted, whether it be circumscribed and of the usual impact or whether it be diffuse and striking. "Irradiation" is a term describing a precordial impulse that spreads over the left chest quite beyond the confines of the overacting heart beneath. Note the *maximum cardiac impulse*; it is usually in the fifth interspace, 8 centimeters or so to the left of the midsternal line, and may be displaced by malformations of the chest, by cardiac affections or by extracardiac conditions (as further noted under percussion). *Epigastric pulsations* and pulsations of the hepatic region come in for mental note; the former might be caused by an enlarged right ventricle, although not often so. The

latter may occur as a result of visceral engorgement, further reflected in distended and tortuous veins. *Reverse the patient*; aneurisms occasionally show pulsations only in the back. Glance at the 10th and 11th interspaces on the left side posterior for systolic retraction—Broadbent's sign of adhesive pericarditis, present when there are extensive adhesions to the diaphragm.

PALPATION.

The palpating hand should be warm. The sense of touch is employed for several purposes, the first of which is the detection of *thrills*. Thrills give a sensation similar to that imparted when the hand is laid on a purring cat. They may be noted (1) in the carotids, one of the signs of aortic stenosis; (2) in the enlarged thyroid, associated with exophthalmic goiter; (3) in the suprasternal notch, due perhaps to an aneurism of the arch, in which event it may also be felt in the second interspace anteriorly, or in the back; (4) and at the apex one may note the presystolic thrill of mitral stenosis or at the base detect the systolic thrill of aortic stenosis. *Hard pressure may obliterate a thrill*. The palm of the hand will more often detect it than will the finger-tips.

Precordial thrills are not always significant of heart lesions. Hearts which are overacting either because the patient is of a neurasthenic temperament or as a result of the excitement or emotion incident to examination, may exhibit a forcible precordial impulse which has all the vibratory characteristics of a coarse thrill. Systolic thrills at the apex of the heart are very rarely of pathologic significance.

Palpate the thyroid to determine that it is an actual enlargement of this gland that rises between the fingers when the patient is told to swallow. The physician may be misled by possible lymphatic enlargements in the neighborhood of the thyroid or by thickly-developed sternocleidomastoid muscles that confuse the picture. Accessory thyroid tissue may occasionally be felt in the suprasternal notch. This is an opportune time to develop the *tracheal tug* (Oliver's sign), by slightly elevating the head and gently grasping the trachea between thumb and finger to note a faint, gentle pull as the heart pulsates—due to aneurism of the arch of the aorta. Cutaneous hyperesthesia or *tenderness* of the pectoral muscles may now be sought for—a symptom quite frequently present in neurocirculatory asthenia—by gently grasping the border of the pectoralis major muscle, the patient's attention being distracted by engaging him in conversation. At this time apical tenderness which is sometimes present in mitral stenosis, may be noted.

To locate the *maximum cardiac impulse*, (sometimes called the point of maximum intensity), is often difficult in a heart with a diffuse, irradiant impulse. It is desirable to locate it for purposes of mensuration and auscultation, and also because the *furthest* point at which the maximum impulse can be felt from the midsternal line is a point that defines the left cardiac border. In some instances I have found it helpful to place the palm of the right hand over the nipple and then bend the second finger under the palm, to touch the chest at that point where the palm feels the impulse *at its maximum*. This point should then be dotted with a blue skin pencil.

The sense of touch further notes, on rare occasions, a pulsating liver. It elicits the presence of abdominal aneurism by a maneuver in which the patient is placed in the genupectoral position and the abdomen palpated; other abdominal tumors change their position, but aneurisms of the aorta remain stationary.

Palpation can be made to yield a wealth of cardiovascular information when it is directed to the pulse—a subject of sufficient importance to warrant its consideration in a separate chapter.

CHAPTER VI.

Examination of the Patient (*Continued*).

PALPATION OF THE PULSE.

AN Iconoclast once remarked to the writer that there were five cardinal points to be observed when visiting the sick; ask where the pain is, how the bowels are, whether the patient slept, look at the tongue—and then feel the pulse long enough to make up your mind what might be the next best question to ask.

REASONS FOR FEELING THE PULSE.

Nowadays, thanks to the revelations of graphic records as interpreted by Sir James Mackenzie and Thomas Lewis, the pulse is felt for many other reasons than sparring for time. It tells the physician whether the heart be regular as to rate, rhythm and volume and permits him to estimate the condition of the arteries. Departures from natural standards put him at once on the track of several cardiovascular conditions, as set forth in Chapter XIII, under “The Irregular Pulse.”

TECHNIQUE.

Three fingers may be conveniently employed in examining the radial artery. The purpose of that finger nearest the patient's hand is to compress the artery in event of a possible impulse reaching the middle finger through anastomosis or deep palmar arch. The middle finger is really the palpating finger;

while the third—that farthest up the wrist—is used in making the gradually increasing pressure which obliterates the pulse-wave and thus permits the estimate of pulse volume.

BIMANUAL ESTIMATES.

In studying the pulse it is well to acquire a set method of examination, so as not to miss information that lies at the fingertips. Therefore the first maneuver is to palpate both radials at the same time, in order to thus appreciate delay or *retardation* of one pulse when compared with the other. This happens, for example, in certain aneurisms of the aorta, or in aneurism of the innominate artery, where the right radial pulse is feeble or absent. Incidentally, the examiner may discover that one radial is a much better pulse to study than is the other, which may lie so deep that its impulse is very faint, or which may be quite absent owing to old injuries of the arm or wrist. Before leaving the wrist, run the finger along each radial to ascertain whether the arterial walls be straight and of the usual elasticity of health, or whether they be resistant, infiltrated, thickened, sclerosed or “beaded”—all of which are degrees of arterial change.

Now place the palm of one hand over the heart, with the other still at the wrist. It will of course be noticed that the radial wave rises one-tenth of a second later than the ventricular contraction, but the *dominant* question should be: “Is every beat of the ventricle accounted for by a pulsation at the wrist?” If not, there is a *pulse-deficit*, caused by (1) premature contractions which rapidly succeed upon the previous

normal cardiac cycle and which, therefore, contract on too small a volume of blood to lift the aortic cusps. These can be recognized by the compensatory pause which follows the premature beat; also by the fact that they will disappear when the heart is accelerated. (2) Or the pulse deficit may be caused by auricular fibrillation, in which event the deficit will become more marked following exercise. The pulse never gives more pulsations at the wrist than occur at the ventricle, although the two waves of a dicrotic pulse may suggest such an event to the uninitiated.

There is still another observation to be made while feeling precordium and wrist. Do any beats seem to completely drop out of their anticipated place in the rhythm of the pulse—and are the identical beats dropped at the heart? If so, an unusual condition has been detected, *viz*: the actual dropped beat. This is a low-grade heart-block, and is capable of eventuating into block of higher grade. Dropped beats must not be confused with premature contractions; the latter occur in *advance* of the anticipated interval.

PULSE-RATE.

There are two extremes of circumstance under which the usual pulse-rate of an individual cannot be obtained. One is when the pulse is quickened by emotion, exercise or excitement; the other is when the individual is in bed, relaxed in body and at mental ease. There is middle ground between these extremes when an average should be drawn. In a previous article¹ I ventured the opinion, based on an analysis

¹ Smith, S. Calvin: An Analysis of Government Cardiovascular Examinations; Jour. Am. Med. Assn., Mar. 30, 1918; vol. lxx, pp. 911-914.

of records, that the natural pulse-rate of active youths between twenty and thirty years of age was higher by ten or more beats than the traditional average of 72 beats per minute. I have since further satisfied myself of this fact by estimating the pulse-rate of 400 young men enlisted in the medical corps of a base hospital. These men had easily passed entrance physical examinations and were youths of normal activities, splendid physique and apparent health. They were examined a half-hour before their breakfast. The average pulse-rate while standing was 84. Consequently, I cannot see that there is any significance in a rather rapid initial pulse-rate, even though it be ninety, *providing there be a well-balanced response* to the exercise test. There is, however, much significance in a pulse-rate of 50; it should arouse the suspicion of heart-block, which suspicion may deepen into conviction with a persistent rate of 40, and quite into assurance when the rate is 35 or less. Bradycardia is also encountered in brain tumor, meningitis, jaundice, in convalescence from typhoid fever and in aortic stenosis.

It is convenient to estimate rate in 5-second counts for a period of 20 seconds. In the presence of any irregularity use full minute counts. The 5-second grouping permits one yet unfamiliar with variation in rate to appreciate it in figures; if there be 6 beats in the first five seconds and 8 in the third, for example, manifestly it is a condition that calls for detailed study. Five-second grouping is also a convenience in estimating the return of rate from exercise (see chart, Fig. 9).

Y. N. A. S. S. :

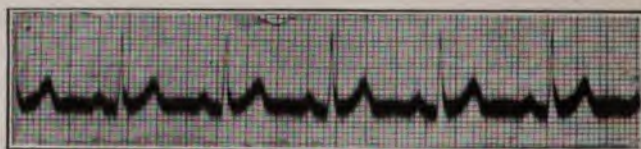
IMPORTANCE OF RATE-RESPONSE TO EXERCISE.

There is one fundamental purpose in examining a heart. That purpose is to *arrive at an estimate of the heart's capacity for work*. This capacity is determined by the efficiency of the *all-essential heart muscle*. The best test yet available for heart-muscle efficiency is its response to exercise. This response is expressed in cardiac and respiratory rates following exercise.

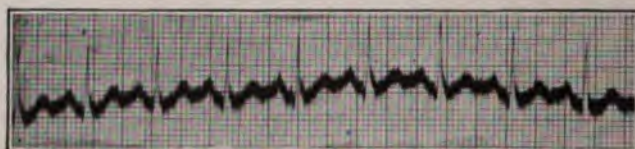
With the above postulate in mind, no heart examination can be considered complete without an exercise test. (Effort will of course be interdicted in the presence of *acute* heart conditions). Many tests have been proposed; none are perfect, for it is difficult to standardize an exercise test. Dumb-bell gymnastics may impose slight effort on a person accustomed to using his shoulder muscles and yet induce prompt fatigue in one who uses his arms but little. Stair climbing is open to the same objection. Hopping on one foot is a form of exercise which is not usually an accustomed practice, and hence is perhaps the least objectionable of exercise tests; but the speed of hopping and the height of each hop should be regulated (see Fig. 10).

The physician may overcome these objections to a degree and attempt to standardize, for his own purposes of comparison, the hopping test, if he will direct that the patient hops on one foot 100 times, with knees slightly flexed at an established angle, keeping count and clearing the floor by about one inch at each hop. It will be necessary to "coach" the patient as he proceeds by saying "A little higher—not

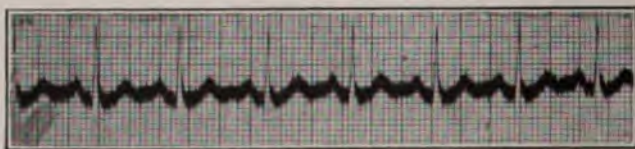
quite so fast—speed up a bit” etc., as the experience of the examiner dictates; for the object is to have



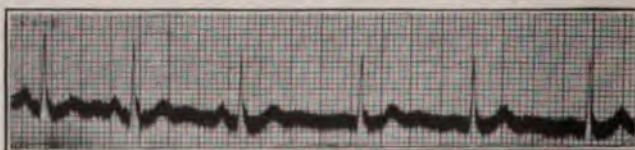
Electrocardiogram from an apparently normal heart before “hopping 100 times on one foot”; ventricular rate, 75; respiratory rate, 16 (Lead II, in this and following figures).



Curve from same heart immediately following 100 hops: ventricular rate, 120; respiratory, 30.



One minute later: ventricular rate, 105.



Two minutes later: ventricular rate, 75; respiratory, 16; both fallen to normal.

FIG. 10.—RATE RESPONSE TO EXERCISE.

The exercise test employed is that of hopping one hundred times on one foot.

this particular patient take just about as much exercise as did the previous patient, in order that the exercise test may more nearly conform to the standard

which the individual physician has adopted as a basis upon which to form his judgment.

WHAT CONSTITUTES A NATURAL RATE RESPONSE?

I have analyzed the pulse-rates of a group of 2215 individuals who were referred for heart examination. Fifty-six per cent. were accepted as having apparently normal hearts. In these the rates per minute were as follows:

ACCEPTED.

Pulse-rate before exercise	Immediately after 100 hops	Two minutes after
91.54	130.13	93.24

Contrast these rates with the rates of the forty-four per cent. who were subsequently rejected on account of valvular diseases, cardiac enlargement, neuro-circulatory asthenia, etc.

REJECTED.

Pulse-rate before exercise	Immediately after 100 hops	Two minutes after
109.16	151.85	121.20

The "accepted" table expresses in an aggregate rates which experience has led me to adopt as quite the usual limits. Following exercise the rate is well-balanced and within 2 minutes approximates the initial rate. Indeed, it often returns within the first 20 or 30 seconds—now for a fleeting interval faster, again slower, then after a few such gradually declining oscillations definitely returning to the pre-exercise rate well within a two-minute time limit. The "re-

jected" table is significant in the *pulse-rapidity immediately following exercise* and in the fact that it *remains persistently elevated* for several minutes afterward. Hurried, panting respirations and an unwarranted degree of physical exhaustion are also the rule in affected hearts. The unaffected hearts of six-year-old children and the hearts of men who are healthy at fifty, respond to the hopping test with a rate-response as natural as that just outlined for young adult life. The child, however, may exhibit a sinus arrhythmia following exercise which was not distinctly noticeable before the test.

There are occasions when it is not wise to submit a patient to the effort of completing 100 hops on one foot. If marked dyspnea be present before exercise or if the initial rate be 140 or over the physician should instruct the patient to exercise only to a point of fatigue, and enter on his records some such observation as "40 hops exhausted patient." In those unable to use their legs or in those of advancing years, resistance exercises of the arms may be substituted to raise the pulse-rate perhaps forty beats higher than the pre-exercise rate, and calculations be thus deduced. An acceleration of approximately forty beats, where obtainable, seems to give the best basis on which to study rate-response.

In private practice, many of the patients who consult the physician are persons who have arrived at the meridian of life and who are frank cardiopaths. In such persons it is often policy to substitute for the more vigorous exercise tests a series of simple bending movements, such as raising the arms over the head and touching the floor ten or fifteen times. The

person accustomed to calisthenics will require many more bending movements than will the one to whom such exercise is a novelty, if the heart rate is to be appreciably raised.

Possible sources of error in rate-response may arise in robust persons, or in others with heart-muscle weariness. A robust person may be so accustomed to vigorous exercise that exercise tests impose no effort whatever on the heart and fails to modify either the pulse-rate or the force of the ventricular contraction. It may be necessary to double the customary exercise when examining such an individual. Again, there are other persons with sluggish or wearied heart muscle—and these are usually individuals of greater weight than one would expect for their height and years—whose pulse-rate is little if any altered by a customary exercise test. In such persons, however, it will be noticed that exercise increases the force of the ventricular contraction, and it is not judicious to exercise them further as heart pain may be thus produced.

It does not minimize the value of an exercise test to remark that it is only one of many signs upon which to base final judgment of a heart. It is to be considered only in its relation to the composite picture of heart affections painted by other carefully elicited symptoms and physical signs.

RHYTHM.

Rhythm is a word that expresses the sense of time. It is measured motion. The healthy heart has a regularity of pulsation whereby the sense of time can anticipate each beat, just as it can anticipate each

tick of a pendulum. Under "Bimanual Estimates" allusion has already been made to the alterations in rhythm occasioned by premature contractions, auricular fibrillation and dropped beats. *Sinus arrhythmia* is perhaps the only variation in pulse-rhythm which is not pathologic; it is quite common in youth and adolescence and is a condition in which the pulse-rate increases on inspiration and decreases on expiration; it is altogether compatible with health. *Coupled* or *tripled* beats, in which the pulse-wave runs along in sets of 2 or 3, disturb the rhythm of the heart and may be due to multiple premature contractions or to an excess of digitalis drugging. In the latter event their occurrence is a signal for withdrawal of the drug. The "5-second count" mentioned under rate as a commendable habit to acquire in pulse examinations, is of splendid service in the study of rhythm. Rate, rhythm and volume should be estimated before and again *after* exercise.

VOLUME.

Volume is that quality of the pulse by which is sensed its fullness or quantity. It is not to be confused with arterial resistance. One speaks of a pulse of "bounding volume" in sthenic fevers; of the "trickling, low-volume" pulse of mitral lesions; of the "thready volume" pulse of exhausting diseases. Under "Pulse Technique" was learned the manner of estimating volume, and finger-tips can usually note any marked deviations in volume that call for the figure-expressed estimates afforded by the blood-pressure apparatus, the sphygmomanometer.

The *regularity* of volume is much disturbed in auricular fibrillation; successive beats do not strike the finger with the same impact; the volume of blood is irregular in its force.

There is an interesting observation to be made on volume before finally leaving the consideration of the pulse. In instances of heart-muscle exhaustion, where the function of contractility is interfered with, each *alternate* pulsation is of lower volume than its predecessor—*pulsus alternans*. It is usually a sign of grave diagnostic import. A convenient bedside maneuver that develops the *pulsus alternans* is accomplished by making gentle pressure on the brachial artery with the disengaged fingers. Gradually increase the pressure to a degree where the weaker of the alternating beats are obliterated and do not therefore reach the wrist. Thus, such brachial pressure produces a sudden cutting in half of the pulse-rate at the radial, a condition obtainable only in *pulsus alternans*.

CHAPTER VII.

Examination of the Patient (*Continued*).

CARDIAC PERCUSSION AND MENSURATION.

PERCUSSION is a term derived from Latin roots and means *to strike through*. It is a method of physical diagnosis devised by R. T. Auenbrugger (1722-1809). There are two forms of percussion, immediate and mediate. Immediate is direct striking of the thoracic wall. Mediate or indirect percussion is the employment of a mediating or intervening substance, usually the finger of a hand, between the chest wall and the percussing finger. Mediate percussion is employed almost exclusively in cardiac examinations; for direct blows over the heart, however gently they may be intended, are not only unwarranted but, as a matter of fact, elicit little information of practical value.

Cardiac percussion is practised to determine the position and diameters of the heart; to establish pulmonary and abdominal conditions which might alter the position of the heart; to ascertain the presence of pericardial effusions; and to elicit any increase in the diameter of the aortic arch. The fact that cardiac percussion is liable to error—principally because many physicians place too much dependence on the *sound* of the percussion-note rather than upon the sense of *resistance* and the actual *feel* of the pulsating organ—should not preclude its thoughtful, systematic employ-

ment in routine examination. The intervening finger should be placed *firmly* on the chest, in a direction parallel with the edge of the organ being percussed; as one acquires experience in cardiac percussion one may prefer to employ the mediate finger in a direction parallel with the *ribs*. Heavy percussion should never be used. The hammer finger should strike a quick, light, elastic blow, not to be often repeated in one spot. Repeated percussion of a given spot only results in confusing the impression gained by two or three well-directed strokes.

Determining the Left Border of the Heart.—Begin at a point on the chest far away from the heart and come toward it. It is well to begin in the axillary line and come forward in the 5th interspace towards the midsternal line, not varying the pleximeter-finger pressure nor altering the percussion stroke. That point at which the note first changes determines *deep cardiac dullness*. As the examiner progresses he will soon elicit *superficial cardiac dullness*, where the air content of intervening lung tissue between the heart and chest wall is considerably less; the pitch of the note, and especially the sense of resistance, is distinctly increased. This point is usually at the outer limit of the maximum cardiac impulse (the “apex beat,” if one chooses to use that term) and is the point which should be marked with a skin pencil as determining the left cardiac border.

Near the junction of the 3d interspace with the left costal cartilage *cardiac flatness* will be elicited; for it is at this point that the heart comes in closest contact with the thoracic wall, without the intervention of lung tissue. This anatomic fact also explains

why a pericardial friction rub is best heard at this area.

The Right Cardiac Border.—Selecting the 4th interspace to the right of the sternum, seek to determine the right cardiac border. Beginning well to the right, approach a point perhaps 3 centimeters from the midsternal line, where dullness may again appear and the sense of resistance be increased. This point determines the right border of the heart—when it can be determined by percussion. It should be routinely sought for, as by the maneuver one may detect malposition of the heart, although it is not always possible to definitely determine the right border. Adding the distances thus obtained from the left and right of the sternum gives the *total transverse diameter* of the heart.

If one desires, one may, by percussion in various interspaces, thus outline the heart upon the chest wall. Completely outlining the heart by percussion may be an interesting employment but not one of especial clinical importance; whether the outline thus obtained would be supposed to represent the heart in systole, when it is much smaller, or in diastole, when it is much larger, would be hard to say (see Fig. 5). The Röntgen ray is more accurate.

The Transverse Arch.—Percussion in the 2d interspace to the right and left of the sternum may elicit an increase in the transverse diameter of the aortic arch, in which situation transverse dullness usually averages $4\frac{1}{2}$ (women) to $5\frac{1}{2}$ (men) centimeters in diameter. Percussion of this area aids in eliminating aneurism, and should therefore be routinely practised. In event of noting by percussion an

increase over the usual diameter, record the findings but suspend definite opinion until confirmed by *x*-ray.

MALPOSITION OF THE HEART.

There are extra-cardiac conditions which may alter the usual position of the heart within the chest. Their possible presence should always be borne in mind when appraising the heart. Such conditions include:

1. Malposition of the patient.
2. Malformation of the chest.
- 3. Spinal deformities.
4. Subdiaphragmatic growths or visceral displacements.
5. Pleural effusions.
6. Extra-pericardial adhesions.
7. Retractive changes in lung tissue.
8. Mediastinal tumors.
9. Dextrocardia.
10. Pericardial effusions.

(1) *Malposition of the Patient.*—It is important that the patient be placed in a correct position for examination. Whether he be sitting or standing, he may hold the muscles of the chest rigid, perhaps through nervousness or apprehension, and thus interfere with the percussion-note and with the *sense of resistance*. By a slouchy or twisted posture he may so depress or rotate the left chest as to change its relation to the underlying viscus. The correct erect position when standing or sitting is with muscles relaxed and arms hanging loosely at sides. If the patient is examined in bed the correct position for the body is flat on the

back; the head may be slightly elevated. It is always inconvenient and sometimes impossible to accurately examine the heart when the patient is twisted to one side or when the chest is flexed from being propped on pillows.

(2) *Malformations of the chest* may alter the relation of the heart to the chest wall. Allowances should be made for any alterations which might be induced by such abnormal chest conformations as the rachitic chest, "pigeon breast," "funnel breast," or "barrel chest," any one of which permits of marked deviations from the position normally occupied by the heart.

(3) *Spinal deformities* have the same effect as have malformations of the chest in displacing the heart. Scoliosis and kypho-scoliosis are more likely to alter the usual position of the heart than are either kyphosis or lordosis.

(4) *Subdiaphragmatic growths or visceral displacements*, such as carcinoma of the stomach, enlargement of the spleen or liver, or an accumulation of fluid in the peritoneum may produce pressure-effects upon the heart's position. Visceroptosis may permit the heart to fall below its customary level. In this connection it may be remarked in passing that the heart has been found altogether below the level of the ribs and occupying the abdominal cavity—*ectopia cordis abdominalis*.

(5) *Pleural effusions*, when left sided, may cause the heart to be pushed far to the right. (See Figs. 11, 12 and 13). An effusion in the right chest may be so extensive as to transmit pressure on the heart and force it to the left, thus giving the impression of cardiac enlargement.



FIG. 11.—CARDIAC DISPLACEMENT (*A*).

The anterior chest wall has been removed. The heart, with its pericardial investment intact, is seen to the right of the midsternal (*M-S*) line. Compare with the two figures which follow.



FIG. 12.—CARDIAC DISPLACEMENT (B).

The pericardial sac has been removed. During life the maximum cardiac impulse was in the 5th interspace to the *right*, 8 cm. from the midsternal line, giving rise to the impression that the heart was completely transposed; for in true dextrocardia one expects to find the impulse as far to the *right* of midsternal line as it is to the *left* of the line in persons who are *naturally* constructed. As a matter of post-mortem fact, however, in this instance the impulse was caused by the *conus arteriosus* (marked *x*)—that conical pouch at the upper and left angle of the right ventricle which communicates with the pulmonary artery. The portion of the left lung which, in the photograph, assumes the position usually occupied by the heart, was hyperresonant on percussion.



FIG. 13.—CARDIAC DISPLACEMENT (C).

The exploring hand of the pathologist detected a resistant mass within the left lung, disposed in an interlobar direction (*i.e.*, upward and backward from the 5th interspace anteriorly). The resistance collapsed under pressure of the hand and 1500 c.c. ($1\frac{1}{4}$ quarts) of pus poured from the left chest. The heart then gravitated toward its usual position, crossing the midsternal line to the degree shown in this illustration. Note the occluding tortuosity of the aorta after the heart has receded, lending color to the ante-mortem statement of the life-long invalid that his heart had "been dislocated ever since an attack of pneumonia thirty years ago." Note also the opaque plaque on the epicardium, which the midsternal line bisects. Such opacities have been called "soldier's spots," for the reason that they are frequently found on the hearts of Civil War veterans; they are probably an evidence of cardio-sclerotic change and are not especially incident to the occupation of soldiering, for they were not observed at necropsies which the writer attended in France.

(6) *Extra-pericardial Adhesions*.—The adhesions which form in pericarditis may be between the parietal and visceral layers; such *intra*-pericardial adhesions do not appreciably affect the position of the heart. When, however, adhesions are *extra-pericardial*, the attachments formed with other structures may so limit the customary excursion of the heart as to constitute a malposition. The heart may thus be attached to the sternum, lungs, diaphragm, ribs or vertebral column.

(7) *Retractive changes in lung tissue*, such as occur in fibroid phthisis, in the right lung may draw the mediastinal structures to the right thus displacing the heart. Or a shrunken left lung may increase the extent of the heart to the left.

(8) *Mediastinal tumors* are among the frequent causes of malposition of the heart. Thoracic aneurisms are quite likely to deflect the organ to some degree, albeit the degree is usually moderate. Cancers or other new growths in the mediastinum may also produce pressure changes in the heart's position.

(9) *Dextrocardia* means a complete transposition of the heart from the left side to the right. It is not to be confused with malpositions which result from left sided pleural effusions nor with retractive changes in lung tissue which displace the heart to the right. Although most medical schools have a case of dextrocardia which can be brought in for exhibition to students, the condition is a rare one. I encountered but three instances of it in two years military experience with hearts, two of the recruits being recognized as medical school specimens. Incidentally, these anomalous men made good soldiers.

(10) *Pericardial effusions* may at times be sufficient to alter the position of the heart, although they more often obscure than alter its position.

MENSURATION.

The heart borders, as determined in the erect posture and before exercise, should be measured and expressed in centimeters from a definite and

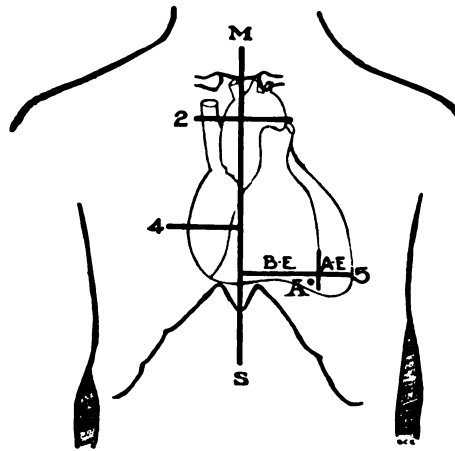


FIG. 14.—CARDIAC MENSURATION.

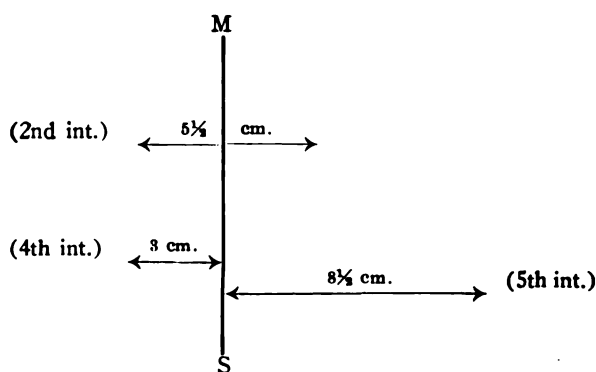
M-S is the midsternal line. *2* represents the second interspace, upon which line is recorded aortic percussion dullness. *4* is the fourth interspace to the right, used for noting any increase in the right cardiac border. *5* is the fifth interspace to the left, upon which is recorded the diameter of the left cardiac border *B-E* before exercise and *A-E* after exercise, as said diameter may then be found to be increased; a healthy heart actually decreases in size following exercise. The dot below line *5*, also designated by the letter *A*, represents the distance of the maximum apical impulse from the midsternal line.

always determinable point on the chest wall, *vis*: the *midsternal line*. This can be marked on the chest with the blued end of a celluoid centimeter rule, drawing it from the base of the suprasternal notch to the

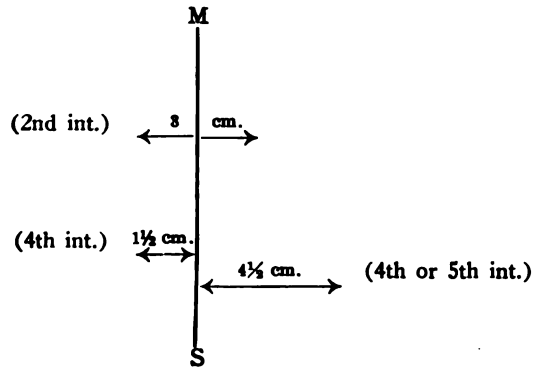
tip of the ensiform cartilage. Such terms as “mid-clavicular line” or “anterior axillary line” or “the nipple line” denote points which are subject to anatomic change or to postural shift. They are indefinite and not always constant, and in cardiac mensuration can well be superseded by the more dependable mid-sternal line. Its convenience, for purposes of case-histories, is illustrated in the diagram superimposed on Fig. 14.

When measuring heart borders, if a flexible rule or tape be employed, it should never be curved in conformity with the chest wall; the reading will be erroneous. The measure should be held straight across the chest for accuracy.

The usual measurements which one would expect to find in health can be set forth as follows—always remembering that the heart diameters which are usual for a robust youth of 150 pounds or more weight, would be most unusual and constitute enlargement in a slender girl of the same age. The figures are the average result obtained from analyzing 1500 cardiac records:—



Healthy children of five years of age or thereabouts have heart diameters which bear a surprisingly constant relation to the above, averaging a little over half of the measurements just given, as follows:—



CHAPTER VIII.

Examination of the Patient (*Continued*).

AUSCULTATION OF THE HEART: MURMURS.

AUSCULTATION of the heart is performed with three purposes in view. First, for the detection of unnatural sounds within the heart or pericardium and to ascertain their relation to the events of the cardiac cycle. Second, to determine the transmission of said adventitious sounds—whether they be propagated along traditional pathways. Third, to elicit alterations in the intensity or duration of other unaltered sounds.

TECHNIQUE.

One may listen to the heart either by applying the ear directly to the chest wall or through the mediation of a stethoscope, an instrument devised by R. H. T. Laënnec in the year 1819, for the conduction of sound. Both physician and patient will prefer the stethoscope to the more intimate method. Placing the ear directly on the chest can be excused in emergency, or when it is desired to estimate auricular activity in a slow heart suggestive of heart-block—for in this condition I have observed that the combined senses of touch and hearing may give information that is not transmitted through the stethoscope alone. The diastolic murmur of aortic insufficiency is also best appreciated with the ear directly on the chest.

As to a choice of instruments, there are many types and each type has its devotees. The writer prefers the instrument known as the "Ford" stethoscope, as simple as it is old, and the property of the medical profession (see Fig. 15). It should be equipped with tubing of sufficient thickness to prevent collapse, of sufficient length to cover the precordium without the assumption of awkward attitudes, and of sufficient pliability to prevent the annoying little cracks in rubber that may admit extraneous sounds. Those who are hard of hearing may choose a make of stethoscope equipped with a diaphragm to intensify sound. Satisfactory auscultation is not so much a matter of stethoscope as it is a matter of *concentration* on the part of the physician, who may have no little difficulty in learning to shut from mind all other sounds than the particular one upon which

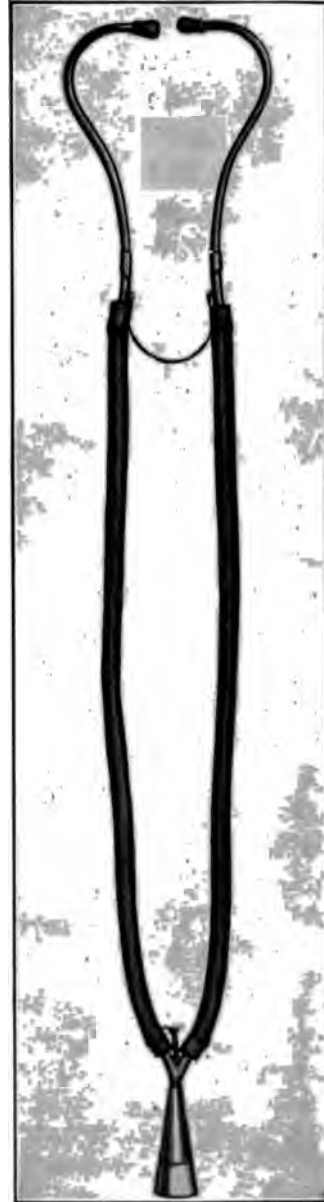


FIG. 15.—THE FORD STETHOSCOPE.

he is then intent. Grading up from these simpler instruments, there are ponderous contraptions of complex design and vaunted theoretical value to place on the precordium, but the choice of instrument resolves itself into a selection to be made between the two types above mentioned. The hard rubber ear pieces should be snugly fitted and comfortable. The best way to secure satisfaction in this matter is to order a selection of various sized tips and try them out until one finds the particular size that makes for clearness of sound, perfect fit and comfort. The stethoscope should be applied firmly to the chest, and it should be evenly applied. Large bells that by their size prevent the entire circumference from snugly fitting in an interspace should be avoided.

NATURAL HEART SOUNDS.

Before taking up the subject of murmurs it will be well to review natural heart sounds and their manner of production. There are two distinct sounds produced at each systole of the heart. *Both the first sound and the second sound can usually be heard at almost any point on the left anterior chest wall.* This sentence is italicized for the reason that an occasional student will imagine that the *first* sound is heard only at the *apex*, and the *second* sound only at the *base* of the heart; as a result, cardiac auscultation is to him confusion worse confounded. There are classical points where the first sound attains *greatest* intensity and points where the second sound is *best* heard—to be described further on under *puncta maxima*.

The first sound is *low pitched, deep seated and prolonged*, in contrast with the second sound which is *higher in pitch, more superficial and short*. The first sound has been compared to that produced by taking the corner of a silk handkerchief in each hand and quickly "snapping" the border taut: this imitates the first sound. Now grasp the border at its middle, and snap only half the length: this imitates the second sound.

The first sound of the heart is believed to be produced by two factors; (*a*) the action of the ventricular muscle, and (*b*) the tautening of the mitral valve curtains, the synchronous action of the tricuspid valve curtains playing a lesser part in the production of the sound. The second sound is believed to result from the closing of the aortic and pulmonary valves.

THE PUNCTA MAXIMA.

There are four classical points (see Fig. 16) on the chest wall where one may hear, with maximum intensity, the natural sounds of the heart. The *aortic* area is at the 2d interspace to the right of the sternum; here the second sound of the heart is distinct. The *tricuspid* area is at the junction of the 5th rib with the sternum on the right, and the louder of the sounds heard here is the first. The *pulmonic* punctum maximum is at the 2d interspace to the left of the sternum; here the second sound is best heard. The *mitral* area is at the 5th interspace to the left of the sternum, and it is at this point that the first sound of the heart is more clearly heard.

While it should be routine practice to listen at these areas, note should be made that *adventitious*

sounds are not necessarily limited to these traditional confines. For example, the diastolic murmur of aortic insufficiency, which is classically located at the aortic area, frequently attains its greatest intensity at the pulmonic area; mitral murmurs often invade the tricuspid area; the systolic bruits incident to intimal roughening of the aorta may sometimes be heard quite generally over the anterior chest wall.

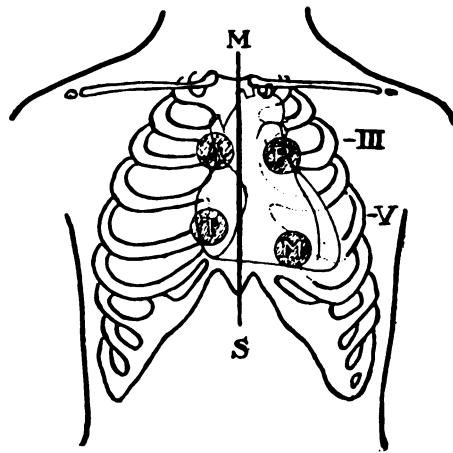


FIG. 16.—THE PUNCTA MAXIMA.

Showing the areas on the anterior chest wall at which the natural valve-sounds are best heard. *A*, aortic area; *T*, tricuspid area; *P*, pulmonic area; *M*, mitral area.

CARDIAC MURMURS.

Definition—A murmur is an adventitious sound which precedes, which takes the place of, or which follows one of the *natural* sounds of the heart. James Hope, who lived 1801-1841, first interpreted many of the adventitious sounds of the heart. The term “bruit” is used interchangeably with the term “murmur.”

Cardiac murmurs are designated first, by their location, whether they be apical or basal; second, by their "time," or place of occurrence in the events of the cardiac cycle. If a murmur occurs during systole (with the lift of the heart) it is a *systolic* murmur; if it occurs after the second sound (during the rest period of the heart) it is a *diastolic* murmur; if it occurs towards the close of this period, just before the systole of the heart, it is a *pre-systolic* murmur.

It is unwise and confusing to time a murmur by feeling a radial pulse, as the radial pulsation occurs one-tenth of a second later than does the systole of the heart. One may make more intelligent use of the carotid artery or, better still, place the disengaged hand over the precordium to time a doubtful murmur.

TONAL PROPERTIES OF MURMURS.

Murmurs may vary in intensity, in quality, in pitch and in duration. Thus the murmur of mitral stenosis varies in *intensity* by gradually increasing in volume of sound as it progresses—this is the "crescendo" element of the sound; the bruit of aortic insufficiency is called "diminuendo," for it decreases in volume of sound as it progresses. *Quality* in a murmur is illustrated by the term "blowing" which is applied to the sound of aortic insufficiency, and by the term "harsh" as applied to the bruit of aortic stenosis. *Pitch* is described by the adjectives "faint" and "loud" as applied to the murmurs of aortic insufficiency and stenosis respectively. The *duration* of a murmur is shown by comparing the *short* bruit of mitral stenosis with the *prolonged* abnormal sound of aortic insufficiency.

The tonal qualities of a murmur—either intensity, quality, pitch or duration—are not safe criterions by which to judge the severity of a valvular lesion.

TRANSMISSION OF MURMURS.

Certain murmurs have classical lines of transmission. The apical systolic murmur of mitral insufficiency is often heard in the axilla or in the left scapular region. Aortic insufficiency, which produces a basal diastolic murmur is frequently accompanied by a Flint murmur at the apex. The basal systolic murmur of aortic stenosis is transmitted to the vessels of the neck. When listening for sound in an artery, undue pressure should not be made with the stethoscope; one thus narrows the lumen of the artery and murmurs may be produced by the onrushing current of blood beneath the point of pressure.

ACCENTUATIONS.

In the presence of valvular disease additional burdens may possibly be thrown on other valves—although it is more probable that the same cause which damaged one valve severely, damaged another to perhaps a lesser degree—and the result is an exaggeration of their usual action and sound, as affected heart structure tries to make up in force what it lacks in efficiency. These exaggerated actions in other valves are called *accentuations*. In mitral stenosis, for example, the second sound, as best heard at the pulmonic area, is accented; the pulmonic second sound is plus, and is expressed by the symbol “P² +.” In the overacting heart of neurasthenia the first sound, as heard at the mitral area, is accented—expressed by

the symbol "M¹ +." In childhood, the second sound of the heart, as heard at the pulmonic area, is usually louder than any other sounds heard at any other areas on the anterior chest wall. This accented pulmonic second sound in children is not of pathologic significance.

REDUPLICATIONS.

One may occasionally notice a tripling of the heart sounds,—a *gallop rhythm* as it is sometimes called. This is not a murmur; when analyzed, the phenomenon is found to be a split first-sound of the heart. It is believed to be caused by the fractionally delayed contraction of one ventricle, which receives the impulse for contraction a fraction of a second later than does its fellow. Clinically, it suggests a blocking of the impulse for contraction in one of the branches of the bundle of His—hence, *bundle branch block*.

FRICTION RUBS.

Friction rubs are occasionally confused with murmurs. The "leathery squeaks" due to *pleural* inflammations can be ruled out of consideration by instructing the patient to hold his breath, when they will disappear. Pericardial friction rubs are not at all constant; they can be made to alter in intensity and at times even made to disappear by shifting the position of the patient.

INFLUENCE OF EXERCISE ON MURMURS.

A *moderate* degree of exercise will *intensify* murmurs that are due to structural valve changes. *Excessive* exercise may "blur out" murmurs that are

faint in pitch or short in duration, by increasing the heart-rate to such a degree that the murmur is blended with a heart sound and cannot be distinguished from it.

A moderate degree of exercise will often cause the *disappearance* of murmurs that are due to relaxed tonicity of heart muscle. So too, a murmur which requires exercise to *produce* it, and which disappears completely when the heart-rate quiets down, *rarely signifies structural valve damage*.

It is well to make a practice of listening to the heart sounds immediately following the routine exercise test, and at short intervals thereafter. In this way one may hear sounds that were inaudible before exercise on account of a thick chest wall or a deep chest.

MURMURS WITHOUT SIGNIFICANCE.

There are many murmurs without significance. Perhaps eight people out of a hundred have them. I kept records of a group of 1940 individuals referred for heart murmurs. Of this number 45.28 per cent were absolutely free from demonstrable signs of heart affections and from symptoms suggestive of circulatory fault. The murmurs were without significance. In this group of auscultatory phenomena, virtually all systolic in time, were (1) *cardio-respiratory murmurs*, occasioned by movements of the heart against residual air in a portion of the lung which overlapped the heart. These were intensified on inspiration, decreased in tone on expiration, and sometimes disappeared when forced coughing probably removed from some bronchiole a mucus plug that had facilitated their pro-

duction. (2) *Postural murmurs*, which seemed to be dependent upon the position of the heart for their production, appearing when the patient was erect, disappearing when he was recumbent. In exceptional instances they were noted in recumbency and disappeared when erect. Others were present in the erect posture, present in the recumbent posture and yet disappeared utterly when the patient was in the genupectoral position. (3) *Tonicity murmurs*, which were present before exercise and which disappeared when exercise increased the tonicity of the heart. (4) *Basal systolic murmurs*, some of which probably had their origin in the *conus arteriosus* of the right ventricle; there were others in which investigation even by the fluoroscope and Wassermann reaction failed to give a clue to their production. (5) *Prolongations of the mitral first sound*, miscalled murmurs.

FEBRILE MURMURS.

Here would seem to be an appropriate place to speak of the systolic murmurs which so often arise during the course of acute infectious diseases. While they call for careful heart watching and while they may be incident to acute endocardial involvement (Chapter XVI), many can be ascribed to no other cause than *relaxed tonicity* of heart muscle; one should not hazard a diagnosis of valvular disease upon the flimsy evidence of murmurs alone. Further, careful physicians often refuse to commit themselves to a diagnosis of valvular disease in a patient who has been examined in the recumbent posture alone.

MURMURS OF DIAGNOSTIC IMPORT.

Basal murmurs, diastolic in time, are of profound significance, and when detected should cause the physician to retrace his steps to elicit additional evidence of heart lesions, which evidence may have escaped him in examination up to this point. The aid of the laboratory should, in addition, be invoked, if there be a suspicion of syphilitic involvement of the aortic valve. *Apical presystolic murmurs* are also most significant, and if presenting with a history of rheumatic fever and accompanied by a snappy mitral first sound, by an accented pulmonic second, by a presystolic thrill, and by an increase in the right heart border the diagnosis of mitral stenosis is justified.

**DIFFERENTIATION OF MURMURS,
FRENCH METHOD.**

There are foreign physicians who employ the oculo-cardiac reflex in the differentiation of murmurs. The *oculo-cardiac reflex* is elicited by making gradual pressure on an eyeball to a point where a change in heart-rate results, due to stimulation of the vagus nerve. The heart-rate may drop from 100 to 60 or even less. It is claimed that under pressure of this degree, murmurs due to structural alterations in heart-valves are *reinforced*, becoming stronger and more distinct, while murmurs due to relaxation of the heart muscle are either *weakened* or *abolished*.

There are circumstances in which this procedure is of value—for instance, when one wishes to reduce the rate of tachycardia in order that one may more clearly discern the heart sounds. It is an age-old physiologic phenomenon, one that is instinctively

brought into play by persons who, in the presence of some horrifying sight, press their hands over their eyeballs, thus in a measure checking the palpitation of the heart which accompanies fright. In some people it is effectual, in others it is not. The French method of examination is at least inconstant and only in an exceptional case yields information which could not be otherwise more definitely obtained.

CONCLUSION.

A murmur, then, should be considered from the following viewpoints: (1) its location; (2) its time; (3) its transmission; (4) its associated accentuations. It should be borne in mind that valvular disease is, for the most part, simply a local expression of a more widely spread condition that at some time involved the all-important heart muscle. With this belief in mind, the physician should finally *correlate the murmur's association with other testimony of heart disease*—such as may be revealed by previous history or by symptoms of early fatigue on effort, precordial pain, cardiac enlargement, visceral congestions, etc., which are the symptoms suggestive of *heart failure* (see page 203).

In the final analysis, if a systolic murmur be the *only obtainable evidence* by which to judge a heart, it should be regarded as irresponsible testimony, which might well be thrown out of court and the case discontinued.

CHAPTER IX.

Laboratory Aids in Diagnosis.

MODERN clinical laboratory methods offer much help to the physician who avails himself of their aid in cardiac affections. They not only confirm diagnoses, but when employed in obscure circulatory faults may establish the presence of conditions which do not present the usual clinical symptoms, perhaps because the primary disease is not yet sufficiently advanced.

It is beyond the purpose of this volume to outline laboratory technique. Nor could such an outline be of much service, for laboratory procedures are always changing as methods improve. A brief reference to those procedures that are of value in the study of cardiovascular conditions will suffice to furnish the cue for their employment.

Urinalysis is routine practice in cardiac as well as in other conditions. It is an index to the metabolism of the body; it furnishes a clue to kidney causes of hypertension; it may reveal permanent damage of renal structure due to an antecedent infection which, for years, has been hampering a heart. The information which urinalysis and the *renal function test* give as to elimination is of profound importance in the treatment of cardiac disease, where free elimination relieves the burden thrown upon the weakened, laboring heart.

It might not be inappropriate to mention at this juncture the *ophthalmoscope*, and its employment in connection with a search for kidney damage which

involves the cardiovascular system. Retinal changes which take place in beginning arteriosclerosis have, in many instances, been the first indication of the approach of that condition. Syphilis, too, may be suggested by eye examination.

Wassermann's serologic reaction should be employed in all involvements of the aortic valve or aortic arch; in arterial thickenings of those whose arteries are infiltrated beyond their years; and in cardiac enlargement of indefinite origin. In obscure cardiac conditions the Wassermann reaction may reveal syphilis as the cause of the malady. Unfortunately, the reason is not always dependable. The test may be negated by alcoholism or by jaundice; it does not always confirm strong clinical evidence of the disease, or even respond positively to a definite history of an initial lesion. Under such circumstances the *colloidal gold test* of spinal fluid should be employed to corroborate the physician's strong clinical suspicion of syphilis.

Differential blood counts may reveal *anemias* that are starving heart muscle as well as other bodily tissues. They may occasionally establish *polycythemia* as the cause of a puzzling and symptom-free cyanosis for which a patient seeks advice. A *lymphocytosis* is strongly indicative of a septic process within the body which may be disturbing the heart action. During the course of low grade fevers or during convalescence, a *leukocytosis* that is otherwise unexplained may suggest the search for myocarditis. *Blood cultures* in malignant endocarditis will identify the organisms from which an autogenous vaccine is desired in the battle with this desperate condition.

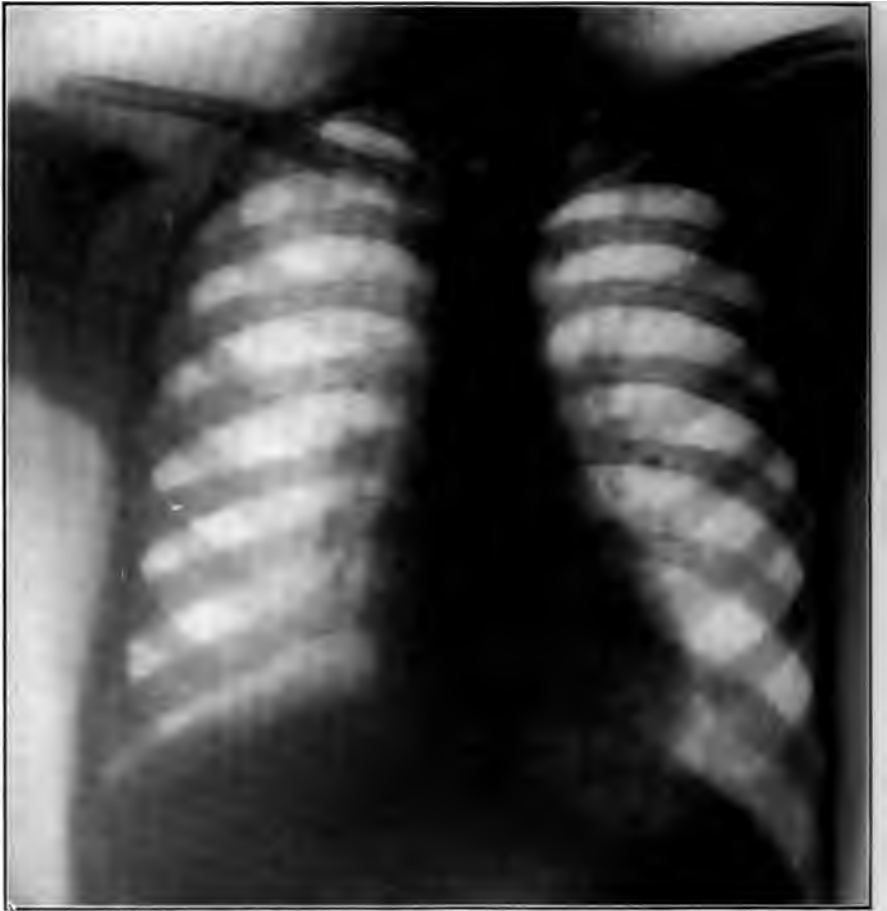


FIG. 17.—THE USUAL OUTLINE OF THE HEART.
As obtained by X-ray examination. (Courtesy of
Dr. Willis F. Manges.)

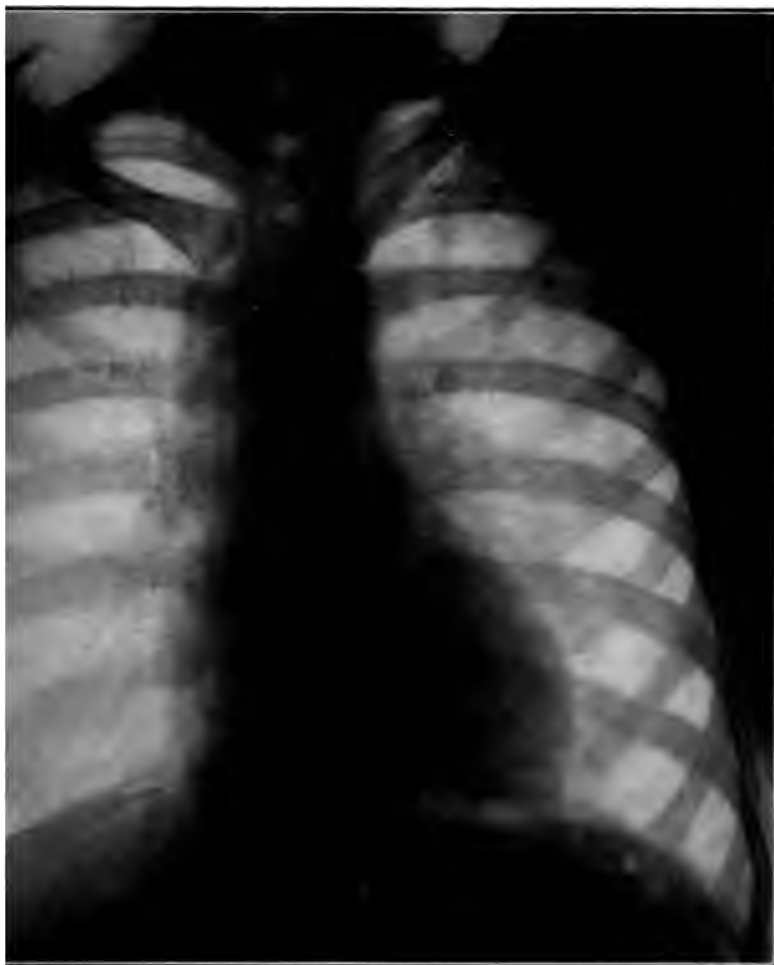


FIG. 18.—CARDIAC ENLARGEMENT.

As revealed by X-ray examination. Compare with Fig. 17.
(Courtesy of *Dr. Willis F. Manges.*)

The *x-ray* may confirm a suspicion of enlargement of the aortic arch. It will detect aneurisms of the thoracic or abdominal aorta. It will define the borders of the heart (Figs. 17 and 18), reveal the presence of fluid in the left chest, locate foreign bodies, and be of infinite value in the search for foci of supuration that cause cardiac disturbances, such as are often revealed by skiagraphy of the apices of teeth.

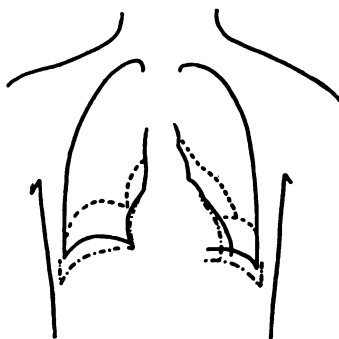


FIG. 19.—EFFECT OF RESPIRATION ON THE HEART.

As obtained by orthodiascopic projection. The solid line shows the heart's position during ordinary respiration; the broken line indicates its position during forced expiration; the dot and dash line shows the effect of deep inspiration. (Clayton and Merrill.)

The *fluoroscope* and *skiagraph* are the most available forms of Röntgen light. The fact that in cardiac mensuration their revelations are less mathematically exact than are the definite measurements obtained from orthodiagraphy should not restrain their employment.

The *orthodiascope* (Fig. 20) is an adaptation of the *x-ray* which outlines the diameters of a viscus by means of rays of light that do not cross each other and thus distort the shadow. Its measurements are



FIG. 20.—GROEDEL ORTHODIASCOPE.

The patient sits with his feet resting comfortably on the adjustable step (*ST*); the side plates (*SP*) can be lowered or raised and pushed in or out; they are placed so that they fit snugly in the axillæ and against the chest of the patient. The fluoroscopic screen (*FN*) is grasped by the finger of the right hand in a small ring (*R*). The bar *B'* carrying the screen is connected by the hollow crossbar (*CB*) with the bar *B*; the latter carries the X-ray tube so that any motion imparted to the fluoroscopic screen by the observer moves the X-ray tube as well. By loosening the nut (*N*) the bar (*B'*) may be slid along the crossbar and the screen may be placed any convenient distance from the patient's chest. *RB*, rubber bulb used for marking purposes; *SC*, sliding counterweights so that the screen and X-ray bars may be properly counterpoised; *C*, cranks used to place the table in the horizontal position if necessary; *LS*, levelling screws; *FSw*, the electrical foot switch. (*Neuhoff*.)

accurate. Manipulation of this apparatus is highly technical, and while some clinicians have acquired skill in orthodiagraphy, more satisfactory results can be secured if operation be left to the skilled Röntgenologist.

CHAPTER X.

Graphic Methods of Examination.

THE POLYGRAM AND ITS INTER- PRETATION.

THE polygraph—from the Greek, meaning many writings—is an instrument which records upon a strip of paper the events transpiring in the “right heart” and in the “left heart.” It is of infinite value in the study of pulse irregularities and sheds light upon cardiac conditions otherwise obscure. It is second only to the electrocardiograph in the information which it reveals, and possesses the advantages over that complex apparatus of (1) portability; (2) bedside convenience; (3) initial low cost (comparatively) with no maintenance expense. Further, it requires no special training for its successful manipulation, other than the native gifts of patience, continuity and attention to small details. Polygraphy is a fascinating as well as a most instructive study.

THE APPARATUS.

The Mackenzie ink polygraph is the apparatus described and referred to throughout this article. It consists of a clock-work mechanism, which operates a time marker and feeds a strip of paper at chosen speeds; of tambors, which permit excursions of the recording inked pens; of a wrist appliance and tambor, of a precordial tambor, and of a jugular cup,

the three last mentioned being connected with the recording apparatus by rubber tubing (see Fig. 21).

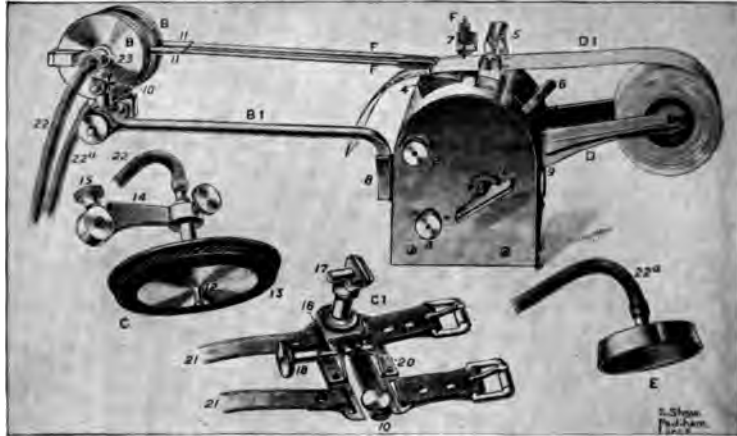


FIG. 21.—MACKENZIE'S INK POLYGRAPH.

A, Clock mechanism. *B*, Pen tambors. *C*, Wrist tambor. *C1*, Wrist appliance. *D*, Paper roll and holder. *E*, Receiving cup. *F*, Writing pens.



FIG. 22.—POLYGRAM OF AN APPARENTLY NORMAL HEART.

Heart was beating tranquilly, uninfluenced by emotion or disease. The vertical lines at the end of the record are *ordinates*. The top record is made by the time-marker, each interval measuring 0.2 of a second. The middle record is the phlebogram, made from the jugular bulb. The lowest line is the arteriogram made from the radial artery.

COMPONENT PARTS OF THE TRACING.

The tracing made by the polygraph is called a *polygram* (see Fig. 22). The component parts of a

polygram are: (1) a *time-record*, which is spaced at intervals of two-tenths of a second, essential in analysis and when comparing the events transpiring in right and left heart; (2) *the arteriogram*, which records the events of the left heart. It is most conveniently recorded from the wrist, and is sometimes called a

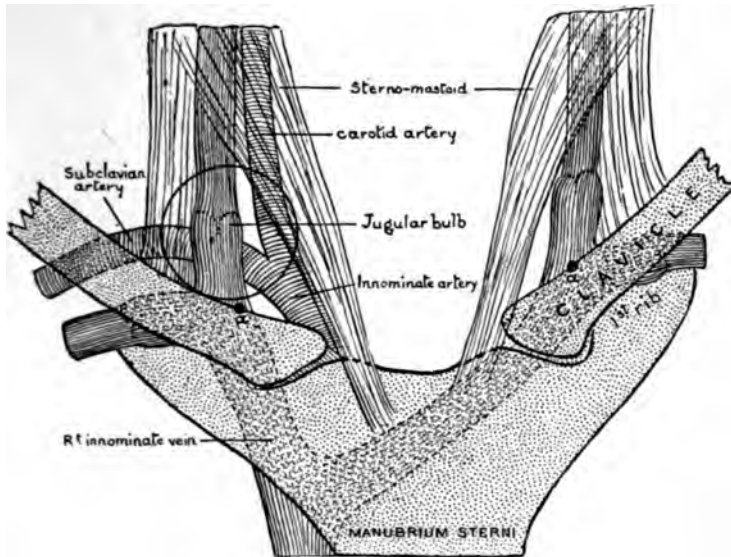


FIG. 23.—THE JUGULAR BULB.

The circle indicates the position for the jugular receiving cup. The spot *a* is one inch from the sterno-clavicular articulation. (*Keith.*)

sphygmogram. If one obtains this record from the tip of the left ventricle it is called a *cardiogram*; (3) the *phlebogram*, which records the events of the right heart. It is most conveniently recorded from the jugular bulb, which is one inch from the sterno-clavicular articulation (Fig. 23). On rare occasions one may obtain it by placing the receiving-cup over a pulsating liver. (4) The *ordinates* from which to

measure. Ordinates should be routinely put in all tracings at 4- or 6-inch intervals by stopping the mechanism and flicking the recording pens with the finger tip.

TECHNIQUE.

If one would be spared annoyance and irritation, one must acquire system in the technique of pulse tracings. Habitual practice of the following points will save time, trouble and embarrassment, as one sits by the recumbent patient and opens the carrying-case (Figures refer to those on illustration No. 21):—

1. Wind clockwork of paper-feed and of time-marker (*A*, 1 and 2).
2. Release starting lever (6) to see that feed-roll revolves and that the speed-control knurl (3) has not locked itself.
3. See that the time-marker (7) has its customary excursion.
4. Adjust paper-bracket *snugly* in clock-case (9). Paper is to feed from top of the roll and run parallel in feed-guides.
5. Test tubings (22). Close one end, blow in the other, to see if they are airtight.
6. Insert tambor arm *snugly* in clock-case (8).
7. Use tubing to test all three tambors (*B*, *B* and *C*) to see if tambors are air tight.
8. Place time-pen on time-marker (7). Insert the two tambor pens (*F*, *F*) and ink all three. Raise tambor pens slightly from paper.
9. Attach tubing to tambors. Insert jugular cup (22*) in distal end of that tubing which supplies tambor nearest time-marker.
10. Apply the wrist-piece (*C* 1) and attach wrist-tambor (*C*).

The Wrist Piece.—One is well repaid for a little care in applying the wrist piece. The patient should be recumbent, with arm comfortably extended along his body, palm upwards. The wrist should be supported by a Turkish towel, folded several times and placed under the wrist, permitting the hand to fall back and thus bring the artery into prominence. The patient should be instructed to comfortably relax his wrist and not disturb its position or twist his fingers

throughout the seance, as he would thus distort the tracing. The tip of the tambor should now be placed directly over the artery at that point where the radial pulsations are strong and superficial. If it be faultily placed to either side of the artery the arteriogram will be distorted. Adjust the tip so that it presses lightly on the artery; heavy pressure destroys the marking of the pulse-wave—as also does insufficient pressure. Now attach the tubing to the wrist piece, making sure that the “fling” of the pen is of sufficient excursion. Experience will teach what is meant by the “fling” of the pen; it should write in waves of sufficient height, instead of in miserable little points that scarcely leave the base-line.

The Jugular Cup.—The jugular pulsation is obtained by placing the metallic cup over the jugular bulb (one inch from the sterno-clavicular junction and directly above the clavicle) with a moderate degree of pressure, which should be varied until one sees the recording pen fling actively—three times as often as does the radial pen and with greater excursions, usually. Or, the excursions may be small and the waves still be capable of analysis. Any movement of the jugular pen which but imitates the radial pulsation is an indication that the metallic cup is more on the carotid artery than on the jugular bulb (see Fig. 22).

In some patients it may be most difficult to find the jugular pulsation. Especially is this so in the individual who cannot relax, and who keeps his sterno-cleidomastoid muscles tense, thus holding the cup away from the bulb. It may take several minutes to tire out this resistance of the neck muscles. Even in the docile type of person it occasionally requires

much patient search and the adoption of such tactics as the shifting of the patient's head to one side or the other, the bending of it a little forward or backward, or even the placing of a small pillow between the shoulder-blades, in order to bring the bulb more prominently from behind the clavicle. Or, having tried these maneuvers on the right side of the neck, it may be necessary to try them on the left side, in the search for a satisfactory jugular pulse. One should not be disgruntled if, in spite of efforts, the jugular bulb still eludes him; persistence will bring its reward.

Making the Tracings.—All is now in readiness for the final step in preparation,—the putting of the pen-points on the paper strip. Adjust them by manipulating the tambor-heads so that the tracings are properly spaced on the strip and do not fling into each other. The points should press lightly on the paper; heavy pressure makes them drag and shortens their excursion.

The tracing is begun by operating the starting-lever. It is a good habit to make two 6-inch tracings at slow motor-speed—in a slow record alternation of the pulse can be more easily identified. Stop the clockwork, adjust the speed-control and make two at moderate motor speed; repeat the operation at high motor speed—not forgetting that in each 4 or 6 inches of tracing ordinates are to be artificially inserted.

There are of course occasions when one will not care to stop the motor to vary its speed—information can sometimes be better obtained from an uninterrupted tracing—as, for example, when searching for a pulse-irregularity which occurs only at exceptional intervals or when studying grades of heart-block.

THE NORMAL ARTERIOGRAM.

Each radial impulse, as recorded in the arteriogram, consists of the *percussion wave* (*p*), followed by the *dicrotic notch* (*d*) after which occurs the *tidal wave* (*t*). From the beginning of *p* to the base of the dicrotic notch, represents the systole of the heart. Diastole occupies the time represented between the base of the dicrotic notch and the rise of the percussion wave of the following cycle. The intervals between beats are evenly spaced; the percussion-waves are of the same height; the base-line of the tracing is uninterrupted. This constitutes a normal arteriogram.

THE NORMAL PHLEBOGRAM.

The jugular pulsations, as recorded in the phlebogram, consist of first, the *a* wave, which is due to auricular contraction. Second, the *c* wave, which is produced by carotid pulsation and by ventricular systole; it might really be called a ventricular wave. Third, the *v* wave (it is in fact a stasis-wave), produced by a filled-up auricle during ventricular systole.

Identifying the a-c-v Waves.—From the ordinate on the right of the radial tracing measure with dividers to the beginning of any percussion-stroke. Carry this distance to the time-marker and reduce it by one-tenth second. Carry said reduction to the jugular tracing; place one point of the dividers on the jugular ordinate. The point of the other divider will now denote the *c* wave. Mark it *c* on the tracing.

Return to the same percussion-stroke in the radial tracing. Measure from its rise to the base of the dicrotic notch. Carry this distance to the jugular

tracing, placing the left point of the dividers where the *c* wave begins. The other divider now points to the *v* wave. Mark it *v* on the tracing.

Return a second time to the first selected percussion-stroke of the radial tracing. Measure the distance to the following percussion-stroke. Carry this distance to the jugular tracing. Place left point of dividers on beginning of *c* wave; the right point now rests on the *c* wave of the *following* cardiac cycle, which is marked *c*. Place the same distance on the beginning of the *v* wave; the right point will then rest on the *v* wave of the same following cycle. Return to the arteriogram, select the following cycle, and repeat the above performance until several *c* and *v* waves are thus identified.

There will now be noticed between the *c* and *v* waves of the jugular tracing which has been marked, another wave immediately preceding the *c*, as yet unaccounted for. It is the *a* wave and occurs at regular intervals. It is to be marked *a*. The jugular tracing should now show a succession of regularly recurring *a-c-v* waves.

Variations in Jugular Waves.—Confusion in the jugular waves may arise (1) through the interpolation of a wave, especially in slow pulses, just before the *a* wave. It is the *h* wave, described by and named after Hirschfelder. (2) Through split waves; *a*, *c* and *v* may exceptionally, each or all, be split. So it would be possible (though not at all probable) to find seven waves in a jugular tracing—three double-splits and an *h*. (3) Quite frequently the *a* wave is piled on the *c*, and may appear as a scarcely-perceptible notch. Here the value of a tracing made at fast motor speed

is manifest; to some extent it spreads out the waves, facilitating their identification.

INTERPRETING THE POLYGRAM.

By the radial tracing can be identified sinus arrhythmia, premature contractions, alternation of the pulse and auricular fibrillation (Figs. 24, 25, etc.). With the aid of the jugular tracing coincidentally considered can be confirmed: (1) auricular fibrillation, by the absence of the *a* wave (in gross fibrillation there may occur fine, fibrillary waves in the jugular); (2) heart-block is established, through the presence of an excess of *a* waves regularly recurring (Fig. 27). Considering the jugular tracing by itself, it shows us delayed conduction, as manifested in the length of the *a-c* interval. This interval, normally between 0.12 and 0.18 of a second, is estimated from the beginning of the *a* to the beginning of the *c* wave. If over 0.2 of a second, conduction is delayed.

THE INTERPRETATION OF ARTERIOGRAMS.

The following systematic steps are good routine to follow when one is ready to analyze the tracing:

1. Look for artefacts in the record, as produced by changes in motor speed, by interruption of the instrument or by the patient moving or coughing. Check-mark and disregard any such artefacts found.
2. Determine pulse-rate by measuring 30 intervals on the time-marker. These equal 6 seconds. Multiply the number of beats occurring in this distance by ten to get the rate per minute.
3. Note the general shape of the pulse-wave. If the patient is highly nervous it may be full of fine

fibrillary waves; if the bed has shaken or if the table upon which the polygraph rests is unsteady, the waves will be altered in shape. The directness with which the percussion-wave rises may suggest aortic lesions;

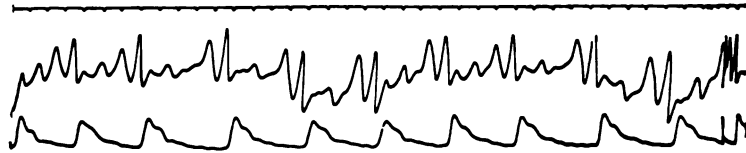


FIG. 24.—SINUS ARRHYTHMIA.

The waves in this and the following polygrams are purposely not lettered, in order that the reader interested in the subject may make his own analyses.

if it has a broad, sustained plateau one thinks of aortic stenosis. The height of the pulse-wave is sometimes an index of pulse-tension.

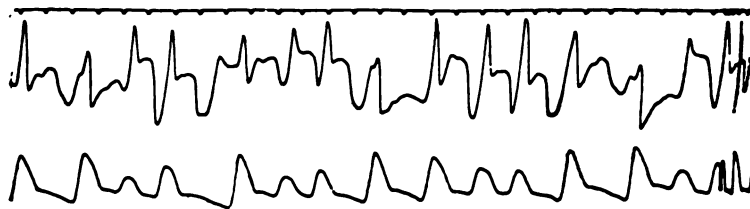


FIG. 25.—AURICULAR FIBRILLATION.

In analyzing this tracing it should be remembered that the *a* wave of the jugular tracing is *absent* in auricular fibrillation, despite the intrusion of an occasional wave suggestive of "*a*" in the phlebogram above. (Courtesy of Dr. Paul D. White.)

4. Determine the presence of a *dominant rhythm*, the rules of which were worked out by Wenckebach.

A dominant rhythm is a fundamental rhythm which governs, more or less, the disordered ventricular movements.

The *presence* of a dominant rhythm indicates either ventricular premature contractions or heart-block. Ventricular premature contractions are recognized by the fact that the distance from the beginning

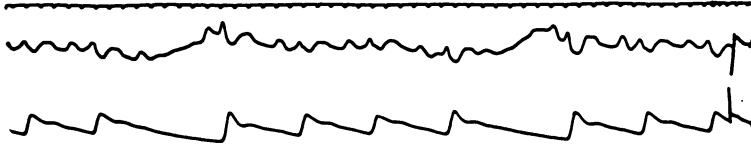


FIG. 26.—DROPPED BEATS.

of the immediately preceding normal cycle, plus the period of disturbance and including its compensatory pause, is equal to the distance of two normal beats. Heart-block is recognized by regularly recurring *a*

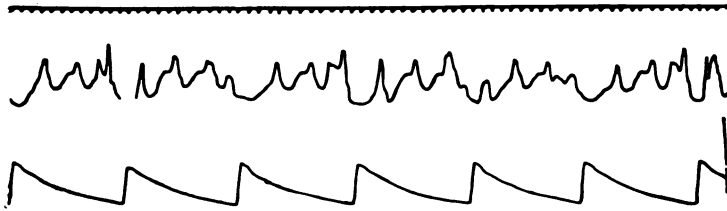


FIG. 27.—COMPLETE HEART-BLOCK.

waves in the phlebogram during the steady fall of protracted radial intervals.

The *absence* of a dominant rhythm indicates auricular premature contractions or else auricular fibrillation. The former condition is measured just as for ventricular premature contractions; but the distance is *not* equal to two normal beats. Auricular fibrillation is recognized by waves that are persistently irregular as to spacing and volume.

Sinus arrhythmia is recognized by the gradual increase of rate during inspiration and an equally gradual decrease during expiration. Inspiration and expiration can often be determined from the general contour of the jugular tracing.

5. When a premature contraction is found, look for alternation of the pulse following it. In alternation each alternate beat is of less volume than its predecessor. Alternation must alternate,—that is, every other beat must be smaller than its predecessor

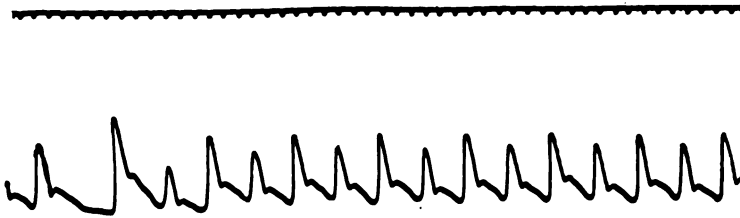


FIG. 28.—PULSUS ALTERNANS.

The alternation follows the occurrence of a premature contraction, and can be more clearly seen if a card is laid over the lower two-thirds of the tracing so as to show only the ends of the percussion waves. (Courtesy of *Dr. Paul D. White.*)

in the radial tracing. There is no such thing as an alternation every third or fourth beat (see Fig. 28).

6. Look for coupling or tripling of the radial waves.

7. Mark the tracing with name and address of patient, date, clinical diagnosis, cardiac diagnosis, polygraphic conclusions.

BRIEF SUGGESTIONS IN ANALYZING POLYGRAMS.

(a) The *a* wave is absent in any weak auricular action—as in auricular flutter or auricular standstill.

(b) Expect to find a split *a* in heart-block.

(c) Sometimes an *a* wave may be seen in the *radial* tracing of heart-block: it is due to the impact of a dilated auricle on the aorta.

(d) A heart-block is called *complete* when the *a-c* interval varies disproportionately in length—as 0.2 then 0.3 then 0.25 of a second, etc.

(e) Any wave that persistently goes below the base-line of the radial tracing is a deep dicrotic notch, and the following wave is a part of the preceding contraction, despite its deceptive height.

(f) Bigeminy is most often due to ventricular premature contractions.

(g) To differentiate bigeminy and alternation: alternation is always late or evenly spaced,—never premature; bigeminy, however, *is* premature.

(h) When a run of regular beats occurs in a grossly irregular polygraphic tracing, *think* of auricular flutter, but confirm the thought by electrocardiography.

Finally, do not try to read too much into a polygram. While it has a splendid clinical use, it is capable of abuse and misinterpretation. This was shown with the first sphygmograph brought to America in 1865, which was discredited because such startling diagnoses as thoracic aneurism were made from radial tracings. Needless to say, the alleged aneurisms failed to appear at necropsies.

CHAPTER XI.

Graphic Methods of Examination (Continued).

THE ELECTROCARDIOGRAM AND ITS INTERPRETATION.

ONE frequently finds in the medical literature of today, illustrations which somewhat resemble the graining in oak, and which are called "Electrocardiograms." The legends which accompany such illustrations are often confusing, for they are written in a language with which many physicians are not yet familiar. It is the intention of this article to explain the principle by which electrocardiograms can be interpreted and their valuable clinical information thus made available to the busy practitioner, who may have had neither time nor opportunity to follow the development of this important subject. Physicians have studied microscopy, urinology, hematology, röntgenology and serology, and considered the time well spent. Electrocardiography, the infant of the laboratory group, is no less worthy of adoption than are its older brothers. And the thorough practitioner should proceed to acquire a working knowledge of this subject, records of which at first glance seem to be only an intricate and unintelligible series of peaks, summits and depressions, but which are, in literal truth, messages from the heart.

DEFINITION.

An electrocardiogram is the product of a modern graphic method of heart examination by which may be differentiated, accurately diagnosed and thus more intelligently treated, various defects of the cardiac mechanism. The first American papers on electrocardiography were not written until 1910, but already the subject has passed the stage of experiment in physiologic laboratories and has become of profound significance to the clinician.

THE PRINCIPLE.

An excitation wave precedes the contraction of the various heart chambers. Kolloker and Müller told us of this in 1856. In discussing the physiology of the heart (page 16) it was stated that this wave normally takes a definite, established route from the pacemaker, where it originates, to its eventual distribution in muscle fibers, which it then excites to contraction. This route is called *the conduction system*.

Now, when disease or its toxins interfere with conduction—when disease disturbs either the rhythm or sequential contraction of the heart; when it enlarges some chamber or renders certain cardiac areas either too highly responsive or too apathetic to the contraction-impulse, the excitation wave will take many devious pathways. By animal experimentation and by clinical experience it has become possible to interpret these deviations in terms of heart affections. The apparatus which records the course of the excitation wave is called an *electro-cardio-graph*.

THE APPARATUS.

Einthoven, a Dutch physiologist, devised in 1903 the instrument by which the action currents of the heart can be led off the surface of the body and made to deflect a very fine gold covered quartz string—a string so fine that the unaided eye can scarcely see it. This wire moves between the poles of a very powerful electromagnet, and is protected from the action of out-

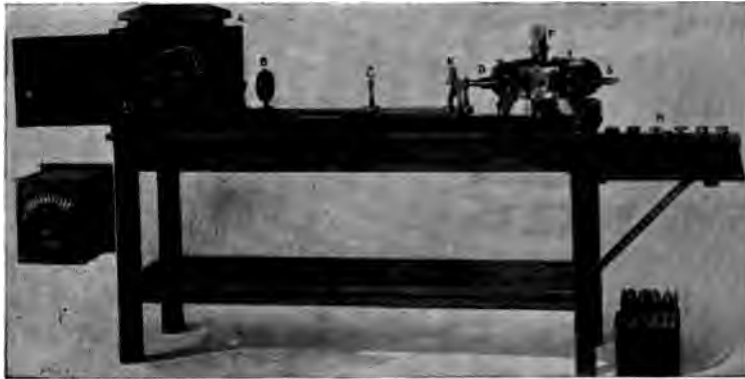


FIG. 29.—THE AMERICAN ELECTROCARDIOGRAPHIC EQUIPMENT.

A is the lamp-hood which encloses the Cunningham arc light; the rays then pass through the condensing chamber at *B*. *C* is a target by which the beam of light is directed on the anterior microscope *D*. *E* is the electromagnet, in the center of which is the string-housing *F*, which protects the delicate string which is actuated by heart-currents. *G* is another microscope for further magnification of the string shadow. *H* is the resistance box which controls the current passing through the string and which protects against outside currents. *K* is the tuning-fork which marks the abscissæ on the electrocardiogram.

side currents by ingenious electrical contrivances. When the string (hence the term “string galvanometer,” which is used as often as is the term “electrocardiograph”) is actuated by the excitation wave, its motions are magnified by microscopes, illuminated by an intense light and the shadow of the oscillating

string is photographed on a moving photographic film. (See illustrations of apparatus, Figs. 29, 30, 31). This procedure is termed *electro-cardi-ography*; the record or *curve* thus made is an *electro-cardio-gram*.

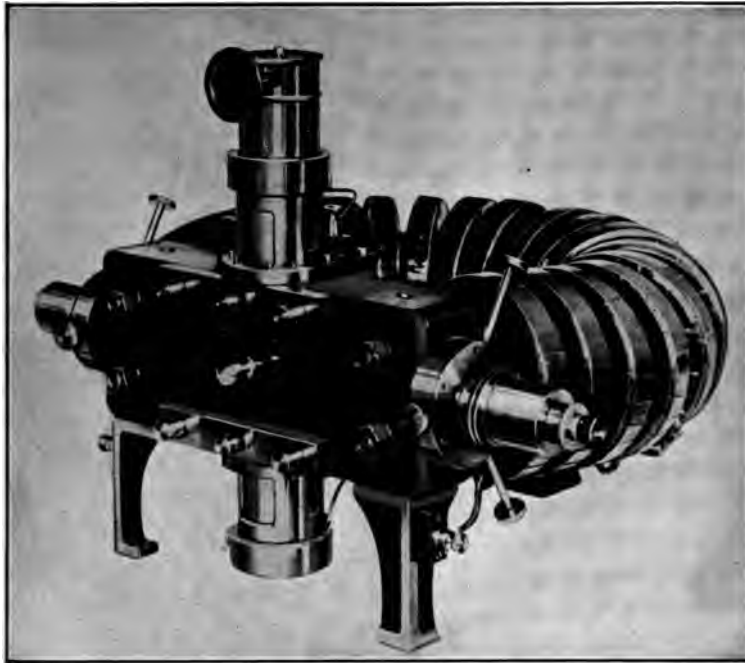


FIG. 30.—THE GALVANOMETER AND STRING-HOUSE.

Closer range, showing how ingeniously the string is protected from particles of dust. The mounting of the microscopes is rigid, yet adjustable.

QUESTIONS THE PHYSICIAN ASKS.

It would burden this chapter to explain at length the elaborate technique incident to the manipulation of the electrocardiograph. Such details may well be left in the hands of technically trained physicians. The clinician is not interested in details of

operation; he is interested in *results*. In order to appraise these results, four questions in connection with this recently introduced clinical aid should be considered, as follows:—

(A) What credentials have established the truths of electrocardiography?

(B) What information of definite clinical value can one expect to receive from the subject?

(C) What constitutes a “normal” electrocardiogram, and how is it read?

(D) What are the more frequently encountered pathologic records with which one should be familiar?

(Answer A.)

Investigations that Established Electrocardiography.

The record from a healthy dog's heart is quite like the record from a healthy human heart; the difference is negligible. In experiments with the dog the conduction system was interfered with at various points by pressure with forceps; it gave abnormal curves. Records similar to these were found in tracings from human hearts; at necropsy in these cases it was discovered that disease had produced the same changes in the human heart that had been experimentally induced in the dog. Another experiment was to produce enlargement of the various chambers in the animal heart; again was obtained a type of curve which had been observed in human tracings; and again did eventual necropsy establish the truth of the electrocardiogram. It was also demonstrated that when infectious disease, such as pneumonia, changed the human records it would alter the records in a similarly diseased dog in like manner. Drugs, such



FIG. 31.—THE CAMERA.

L is the film-roll box; the motor *M* feeds the film past the camera lens *O* at a selected speed, usually at 25 R.P.M. for clinical work. *P*, is a knife which cuts the completed film. *R* is a removable box in which the exposed film drops.

as digitalis, atropine, lead, etc., were administered to canines in the laboratory; the cardiograms were modified. The same alterations in the records were found in patients taking these drugs. With such credentials has electrocardiography introduced itself to the medical profession.

(*Answer B-I.*)

Clinical Diagnoses Confirmed by Electrocardiography.

This method of examination has not only acquainted the physician with heart conditions which were not known to exist before its advent, but it has also confirmed many clinical diagnoses which he has been accustomed to make. For example, it tells which chamber of the heart preponderates in cardiac enlargement. It frequently substantiates the clinical diagnosis of mitral stenosis; it furnishes corroborative written testimony of aortic valvular disease. It guides in the selection of cardiac drugs; it warns of their beginning toxic effects. It not only records the efficiency of chosen methods of treatment, thus often indicating a change of remedies, but it also signals the approach of danger in the overadministration of certain drugs. To illustrate: it tells when the *cardiac tolerance* of digitalis has been reached long before the *physiologic limit* manifests itself. It heralds, at times, the approach of some diseases which affect the heart—such as acute rheumatic fever—before the clinical evidences of fever, joint swellings, etc., make their appearance; and at the further extreme of an illness, graphic records may indicate the approaching end of life long before Cheyne-Stokes respiration and

other traditional symptoms of dissolution ensue. In addition, the study of an extended series of curves, which have been taken under varying conditions of rest and exercise, may afford written evidence of the functional activity of the heart muscle. Structural myocardial change, which is a diagnosis often difficult of clinical determination, can now be recognized by electrocardiography. (See Fig. 43).

(*Answer B-2.*)

Diagnoses Not Possible by Usual Clinical Methods.

Electrocardiography does more than confirm diagnoses at which one has clinically arrived; it gives minute information concerning irregularities of the heart which are clinically suspected but which cannot be certainly sustained without graphic record. Among these are exaggerated sinus arrhythmias, multiple premature contractions, paroxysmal tachycardia of auricular or ventricular origin, atypical auricular fibrillations, auricular flutter, ventricular flutter, varying grades of heart-block, etc.

Such conditions give curves that can be better illustrated than described. Pathologic records are shown on subsequent pages. Now, however, the normal electrocardiogram should be first considered; then one can intelligently study departures from the normal which are induced by disease. It is sufficient to bear in mind, at this stage of the discussion, that disease changes the normal waves of the electrocardiogram in *sequence*, in *amplitude*, in *direction* and in *duration*.

(Answer C-I.)

The Normal Electrocardiogram and How to Read It.

Three curves compose the record; they are the three *Leads*, expressed in Roman numerals (see Fig.

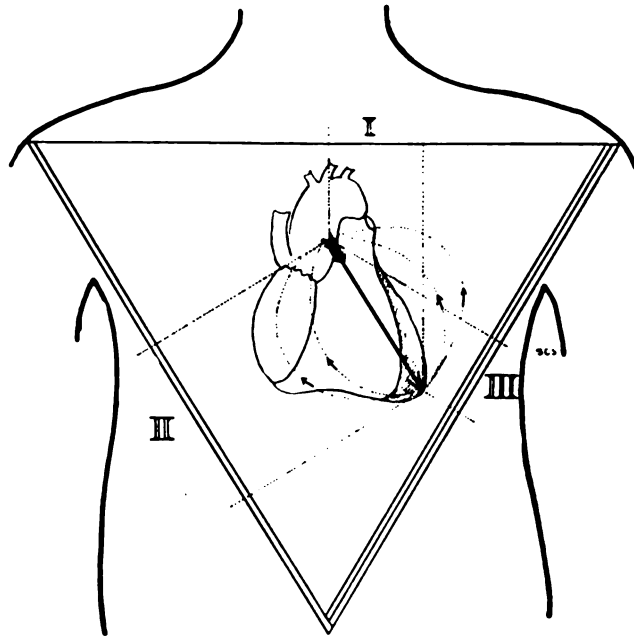


FIG. 32.—THE THREE LEADS.

The heart is represented as within a triangle composed of Leads I, II, and III. The arrow within indicates the electric axis of the heart; the dotted lines represent the direction of the contraction waves from this electric axis. In the normal heart, Lead II registers the greatest value of these contraction waves, for the reason that it is more nearly parallel with said axis, and thus reflects more of the heart's surface.

32). They are called "Leads" because they lead the current from certain surfaces of the heart. Lead I registers the electric potential of that part of the heart which comes nearest to a line drawn from right

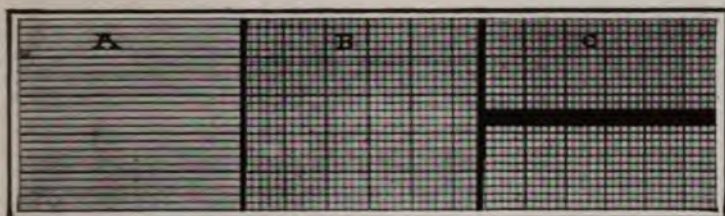


FIG. 33.—COMPONENT PARTS OF THE RECORD.

A shows the ordinates, or horizontal lines, which are engraved on the camera lens. They are used to express in millivolts the amplitude of the various waves. *B* shows the abscissæ, or vertical lines, put on the record by a time-marker; their purpose is to time the various events of the cardiac cycle. *C* shows the shadow of the string when at rest, not activated by heart currents.

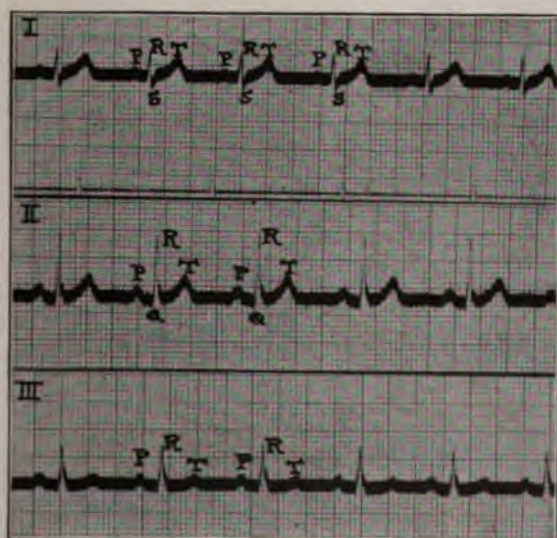


FIG. 34.—NORMAL ELECTROCARDIOGRAM.

P is the representative of auricular contraction. *Q-R-S-T* represents the ventricular complex. The normal limits of the various waves are explained in the text. As Lead II expresses the greatest values of the individual waves, said lead will be used in the pathologic illustrations which follow as a normal standard for comparison.

arm to left arm. Lead II, of a line drawn from right arm to left leg; Lead III, of a line drawn from left arm to left leg. Insulated wire carries the heart current from patient to the string galvanometer. The connection or *electrodes* for the patient are simply pieces of German silver curved to fit right arm, left arm and left leg, and are held in place with bandages soaked in a 20 per cent. hot salt solution.

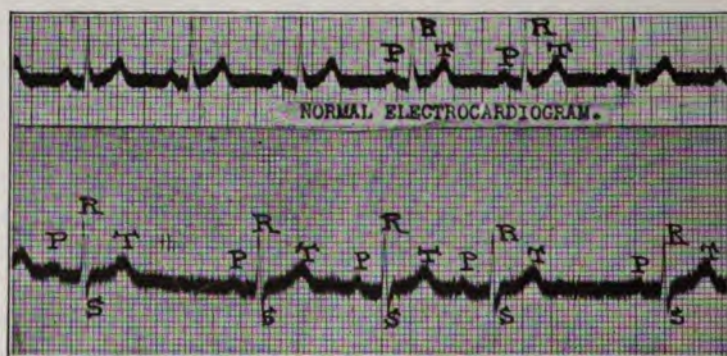


FIG. 35.—SINUS ARRHYTHMIA.

This curve strikingly illustrates the variations in rate which characterize the "youthful type" of cardiac irregularity. The sequence of events is a normal *P-R-S-T* complex, but the rate varies with each contraction. The condition is not of pathologic significance.

The fine horizontal screen lines (see Fig. 33-*a*) are called *ordinates*; they are permanently drawn on the lens of the camera and are used to measure the height of the individual waves, each space being equal to 10^4 millivolts. The vertical screen lines (see Fig. 33-*b*) are called *abscissæ* and are used to time the events of the cardiac cycle, each division representing 0.04 of a second. The *abscissæ* are photographed on the record as shadows of accurately revolving spokes which are regulated by the vibrations of a tuning-

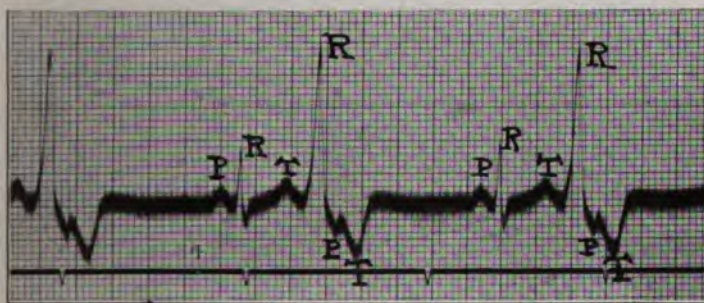
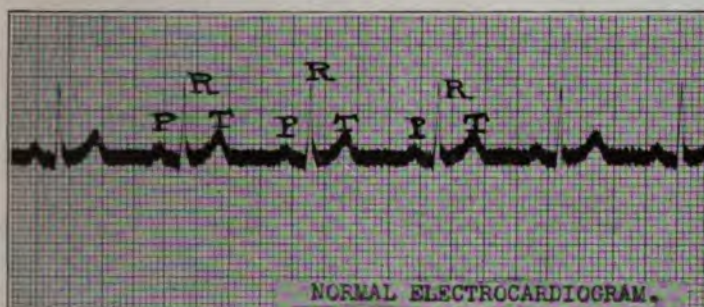


FIG. 36.—RIGHT VENTRICULAR PREMATURE CONTRACTIONS.

Right ventricular premature contractions produced a bigeminal pulse. The premature contractions in this instance are identified by the tall *R* spikes which follow closely on the preceding *T* wave, during what should be the diastolic period of the heart.

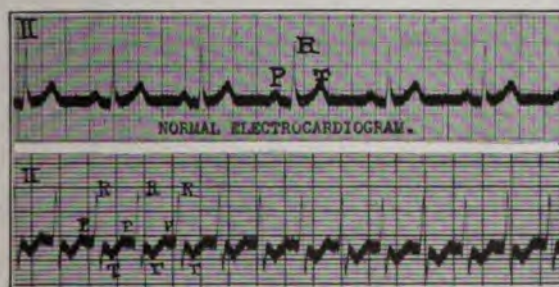


FIG. 37.—PAROXYSMAL TACHYCARDIA.

The ventricles respond to each auricular impulse, both chambers contracting at the rate of 210 per minute.

arm to left arm. Lead II, of a line drawn from right arm to left leg; Lead III, of a line drawn from left arm to left leg. Insulated wire carries the heart current from patient to the string galvanometer. The connection or *electrodes* for the patient are simply pieces of German silver curved to fit right arm, left arm and left leg, and are held in place with bandages soaked in a 20 per cent. hot salt solution.

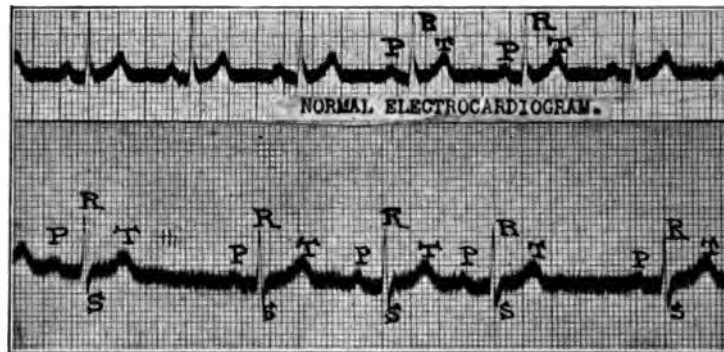


FIG. 35.—SINUS ARRHYTHMIA.

This curve strikingly illustrates the variations in rate which characterize the "youthful type" of cardiac irregularity. The sequence of events is a normal *P-R-S-T* complex, but the rate varies with each contraction. The condition is not of pathologic significance.

The fine horizontal screen lines (see Fig. 33-*a*) are called *ordinates*; they are permanently drawn on the lens of the camera and are used to measure the height of the individual waves, each space being equal to 10^4 millivolts. The vertical screen lines (see Fig. 33-*b*) are called *abscissæ* and are used to time the events of the cardiac cycle, each division representing 0.04 of a second. The *abscissæ* are photographed on the record as shadows of accurately revolving spokes which are regulated by the vibrations of a tuning-

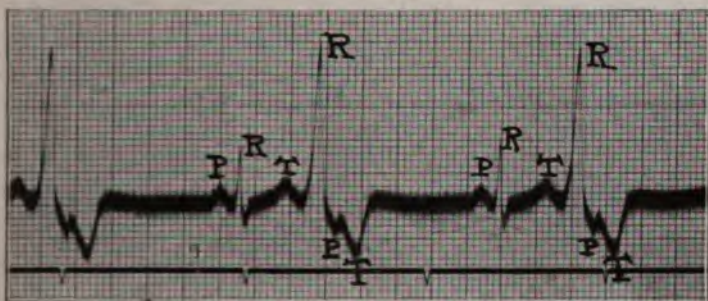
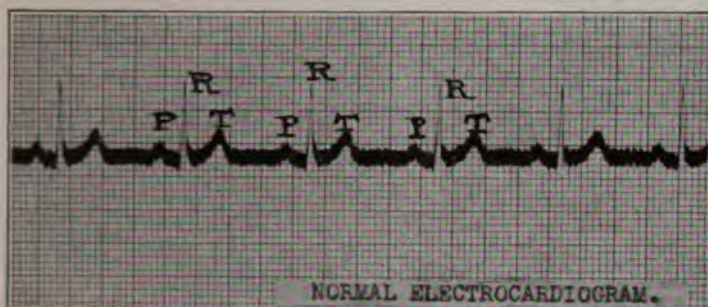


FIG. 36.—RIGHT VENTRICULAR PREMATURE CONTRACTIONS.

Right ventricular premature contractions produced a bigeminal pulse. The premature contractions in this instance are identified by the tall *R* spikes which follow closely on the preceding *T* wave, during what should be the diastolic period of the heart.

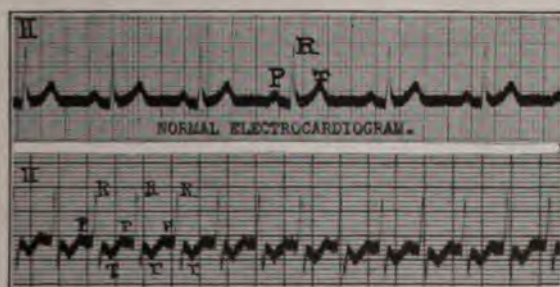


FIG. 37.—PAROXYSMAL TACHYCARDIA.

The ventricles respond to each auricular impulse, both chambers contracting at the rate of 210 per minute.

fork, and called a time marker. Figure 33-c shows the shadow of the string when at rest, not activated by heart currents.

The Symbols of the Electrocardiogram.—Einthoven applied certain symbols (purely arbitrary, with no interpretation to be put on the letters, as would be the case if he had chosen "a" to represent auricular contraction, "v" for ventricular contraction, "d" for

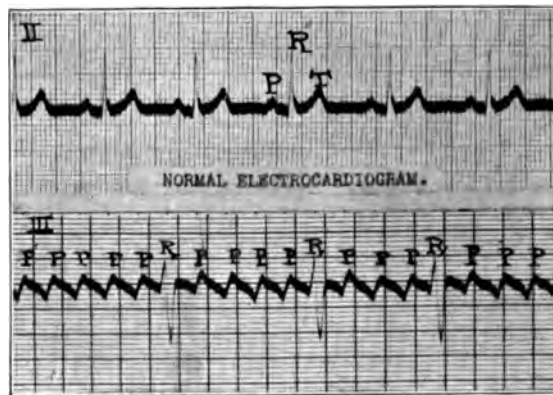


FIG. 38.—AURICULAR FLUTTER.

The auricles are contracting at an average rate of 270 times per minute. The ventricular rate averages 60 per minute. The impossibility of arriving at a clinical diagnosis under such circumstances is quite apparent. Graphic records such as this, however, clearly establish the diagnosis and thus point the way to efficient treatment.

diastole, etc.) to the various peaks, elevations and depressions of the electrocardiogram, which are universally used. Thus the representative of the auricular contraction is called the *P* wave (Fig. 34). It is a small, blunt pointed or rounded elevation, not normally over 2 mm. high nor over 0.02 of a second wide, and is directed upward. If it is more than 3 mm. in amplitude or if it is wider than 0.12 of a second,

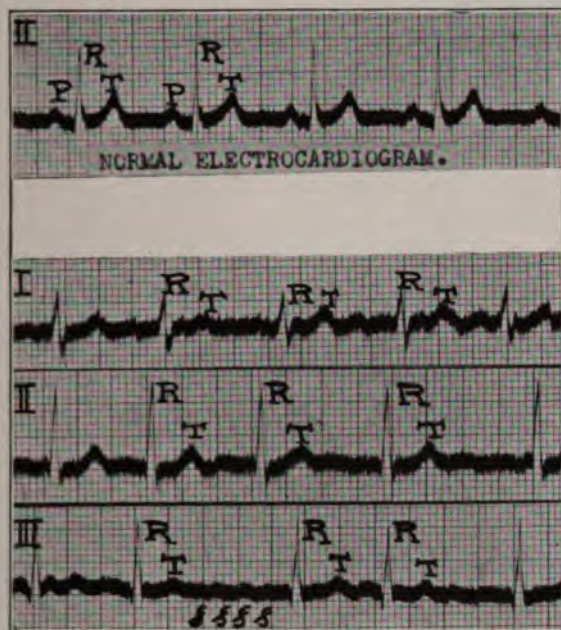


FIG. 39.—AURICULAR FIBRILLATION.

The *P* wave, the representative of auricular contraction, is absent in all three leads. Fine fibrillary ("*f*") waves fill diastole in Lead III. Note the absolute irregularity of ventricular contraction, as expressed in the uneven spacing of the *R* wave.

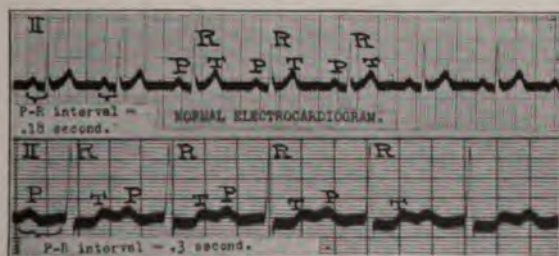


FIG. 40.—DELAYED CONDUCTION.

The impulse for contraction is transmitted from the auricle to the ventricle over the node of Tawara and the bundle of His. In the normal heart this transmission occupies 0.18 of a second. In the lower figure conduction is delayed to 0.3 of a second. Delayed conduction may often be the forerunner of eventual heart-block; hence, such patients require frequent graphic study and appropriate treatment.

it indicates auricular enlargement. *P* may be flat—"isoelectric"—in Lead I, especially if the heart be vertical within the chest.

The time taken for the passage of the impulse from auricle to ventricle is called the *P-R interval*. It is measured from where *P* leaves the base line to where *Q*—or, in the absence of *Q*—to where *R* begins, and normally occupies from 0.12 to 0.18 of a second; over 0.2 of a second indicates a delay in conduction

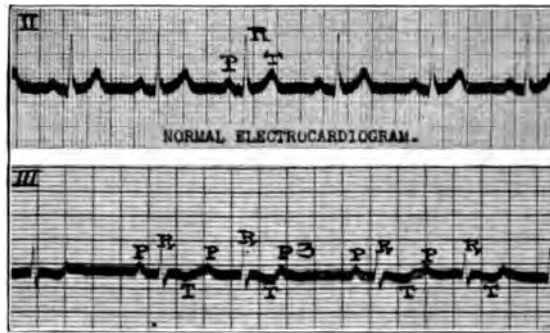


FIG. 41.—DROPPED BEAT.

In the second curve it will be noticed that *P-3* is not followed by a ventricular contraction. *Dropped beat* is in reality a low-grade heart-block, and is not to be confused, as is frequently done clinically, with ventricular premature contractions which fail to reach the wrist.

(Fig. 40). The *P-R* interval in the electrocardiogram represents the same event as does the *a-c* interval in the polygram.

The *Q-R-S-T* complex denotes ventricular contraction. *Q-R-S* indicate the spread of the contraction wave in the fibers of Purkinje. *Q* is the first evidence of activity at the apex of the ventricle; it is directed downward, usually not over 2 mm., and may be entirely lacking, being submerged by stronger contraction

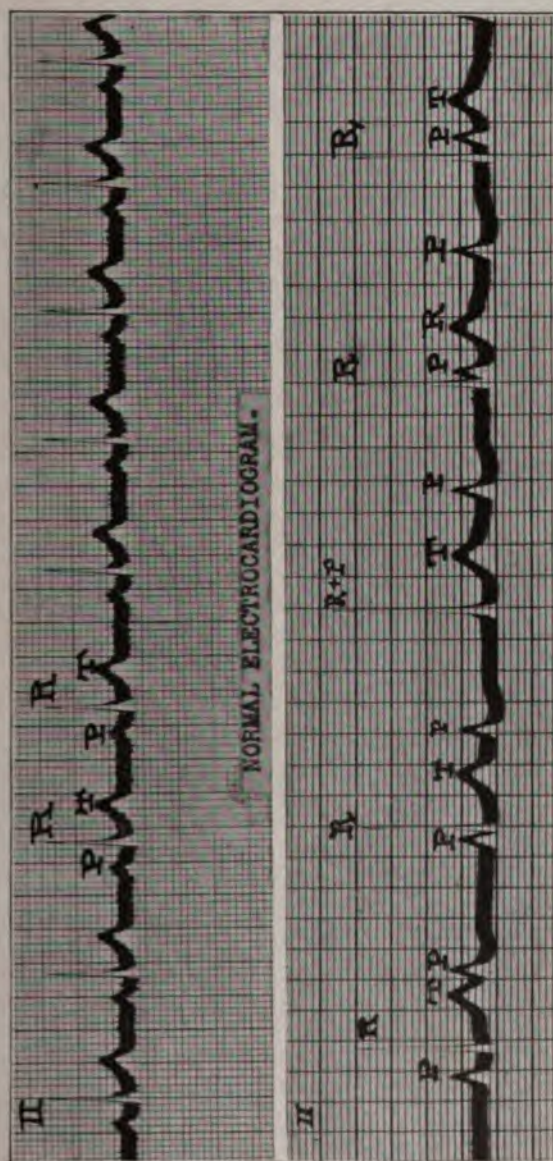


FIG. 42.—HIGH-GRADE HEART-BLOCK.

There is complete dissociation of the auricles and ventricles; while each contracts quite regularly, yet one is absolutely independent of the other. The auricular rate is 90, the ventricular 50 per minute.

waves. The *R* wave is due to the action of the basal portions of both ventricles; it is directed sharply upward in all three Leads, and in Lead II registers its greatest normal amplitude of from 10 to 20 mm. (Usually, in the healthy heart, the values of all waves of Lead II equal the combined values of Leads I and III). The first sound of the heart begins as *R* ap-

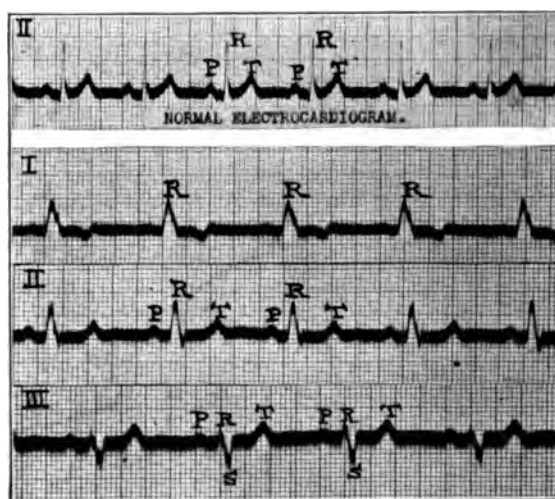


FIG. 43.—ARBORIZATION BLOCK.

The lesion is in the fibers of Purkinje, evidenced by the wide *R* base and by the notched *R* apex.

proaches its zenith. If the distance from where *R* leaves the base-line until it returns be over 0.1 of a second, *R* is said to be *aberrant* or "wandering." The *S* wave is a sharp peak, directed downward, 2 or 4 mm., and is due to activity at the apical portions of the ventricles. When *S* is deep it usually indicates some disturbance in the ventricles. It too, may be lacking, although less often so than *Q*.

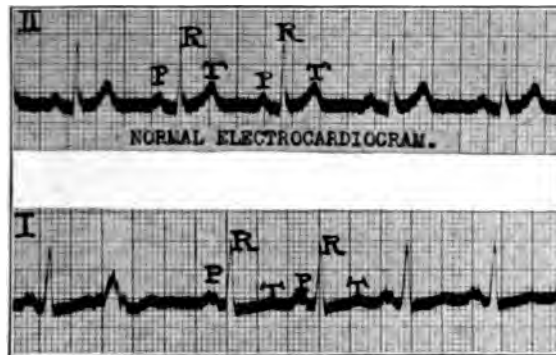


FIG. 44.—FROM A CASE OF MITRAL STENOSIS.

The "split *P* summit" revealed in this curve confirmed the clinical findings of presystolic murmur, snappy second sound and thrill.

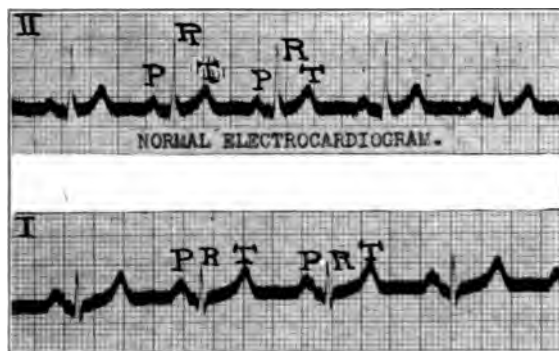


FIG. 45.—AURICULAR ENLARGEMENT.

A casual glance at the above records might suggest that they are from the same patient. A little careful inspection, however, will show that the lower record presents a *P* wave which transcends the normal limits and which indicates auricular increase.

From the end of *S* (or the end of *R*, if *S* be absent) the record is again flat (isoelectric). The potentials in both ventricles are neutralizing each other; hence the return to the base-line or zero. Soon the potential in the right ventricular region begins to pre-

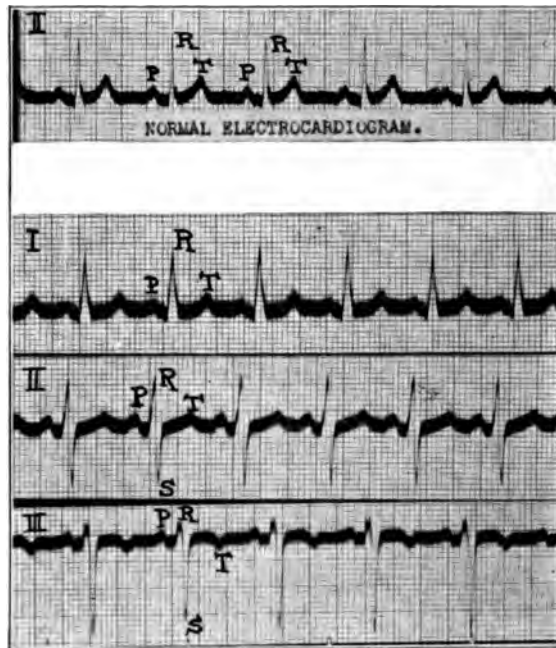


FIG. 46.—LEFT VENTRICULAR PREPONDERANCE IN A CASE OF AORTIC REGURGITATION.

In estimating ventricular preponderance Leads I and III are used for comparison. Note tall *R* in Lead I and the deep *S* in Lead III.

dominate, and a flow of current toward the opposite side of the heart ensues. This current produces the slowly-rising *T* wave.

T represents the final activity of the right ventricle and has its origin, it is believed, at the basal

portion of the ventricle, near the mouth of the great vessel. *T*, as will be noticed, returns to the base-line much more sharply than it left it, due to sudden relaxation of ventricular fibers at the end of the systole. It is normally directed upward (upward direction of a wave is also referred to by the word "*positive*") in all three leads, although it may occasionally be directed downward (*negative*) in Lead III, in which lead it is often quite small. *T* is an interesting wave, and cuts up some peculiar capers: exercise increases it; digitalis flattens or inverts it; changes in the content or in the quantity of the blood make changes in the behavior of *T*. A flattened or inverted *T* is of more pathologic significance than is a high *T* wave. The second heart sound occurs synchronously with the end of *T*.

From *T* to *P* is the diastolic period of the heart. This isoelectric line, due to an absence of potential, is sometimes interrupted by a small *U* wave. There is no satisfactory interpretation of the inconstant *U* wave as yet. It is often accentuated when *T* is accentuated.

(*Answer C-2.*)

Interpreting the Curves.

It is well to get in the habit of adopting a stated method of procedure when analyzing a set of curves. Without system one may overlook important details. The writer has evolved the following order of procedure in considering an electrocardiogram and would suggest its use to those who are beginning to analyze curves.

- GENERAL SURVEY. {
- a.—Are there any artefacts in any of the leads? (An "artefact" is an artificially induced disturbance in the course of a record—due to movements of the patient, to sudden changes in camera-motor speed, to chemical defects in film, etc. Disregard such interruptions).
 - b.—Is the pulse-rate regular or irregular? (With a pair of dividers measure the distances between successive *P* waves and *R* waves).
 - c.—What is the pulse-rate? (Count 30 abscissæ and multiply number of cycles therein by 10).
- SECOND LEAD READING. {
- (The second Lead is the most important of any of the three Leads, individually considered, and gives the most information. It is not wise, however, to commit one's self to an interpretation based on reading one Lead alone, without having compared all three Leads).
 - d.—Is each *P-R-T* sequence normal?
 - e.—Does the *P* wave show any deviations?
 - f.—How long is the *P-R* interval, and is it constant?
 - g.—Does the *R* wave show any deviations?
 - h.—Does the *T* wave show any deviations?
- COMPARATIVE READING. {
- i.—In all three leads: does low amplitude of the waves express low state of contraction-impulse?
 - j.—Comparison of Leads I and III with each other. For example, left ventricular preponderance shoots the (usually tall) *R* spikes away from each other in Leads I and III; so does right bundle-branch block. The differentiation is made by right bundle-branch block showing a *wide R*, which is followed by a *diphasic* (meaning "in two directions") curve. On the other hand, in *right* ventricular preponderance and *left* bundle-branch block the *R* spikes *converge* in Leads I and III.
 - k.—Interpretation.

(Answer D.)

The More Frequent Types of Pathologic Records.

It has been shown what constitute the normal limits of waves. It is now in order to consider the

changes wrought in a normal record by abnormal conditions, looking for departures in *sequence*, in *amplitude*, in *direction* and in *duration* of the waves in Figures 35 to 46 inclusive. For the convenience of the reader in comparative studying, a normal Lead II surmounts each abnormal record.

BRIEF SUGGESTIONS IN ANALYZING ELECTROCARDIOGRAMS

(1) Dextrocardia is diagnosed by finding all the waves including *P* inverted in Lead I.

(2) In looking for a premature beat, examine Lead II: if it occurs in the right ventricle the *R* wave is premature, usually notched and positive (*i.e.*, directed upward); a left ventricular premature beat is *negative* in Lead II.

(3) Left ventricular premature contractions are probably apical in origin; right ventricular premature contractions are probably basal in origin.

(4) Ventricular premature contractions occur six times oftener than those of auricular origin, and are the frequent cause of a bigeminal or trigeminal pulse.

(5) Premature auricular contractions are identified by the *P* wave being a different shape than normal: it not only occurs in advance of other *P* waves in the record, but in shape it may be diphasic, inverted or upright, and *the succeeding ventricular complex is normal*. Again, the *P* may not be altered, but the entire beat be unquestionably premature; this is also due to a premature auricular contraction.

(6) In gauging the *P-R* interval, look for a *P* that begins at an abscissa.

(7) In heart-block always estimate the number of auricular contractions and the number of ventricular contractions to establish the relationship (such as 2 to 1 block, etc.).

(8) Do not make a diagnosis of left ventricular premature contraction and right bundle-branch block in the same record; it is either one or the other.

(9) Auricular flutter is present when the auricular rate is 200 or over and the ventricular complex normal.

THE USE AND ABUSE OF ELECTROCARDIOGRAPHY.

Electrocardiography is not to be used to the exclusion of clinical observations; it will, however, verify and corroborate data thus secured. It will clear up many doubtful diagnoses. It has, further, taught us of many serious heart conditions that cannot be distinctly recognized, and hence not intelligently treated, without its employment. The electrocardiograph, when properly used, is an instrument of scientific precision that is of profound value—and in some instances indispensable—in the study of heart affections.

A single normal electrocardiogram does not invariably mean that the heart is normal. In my electrocardiographic studies of the acute infections of childhood, at the Philadelphia Hospital for Contagious Diseases, I have repeatedly seen electrocardiograms from the same patient that were well within physiologic limits at one examination and distinctly abnormal a few hours later. The reason is clear: toxins may be set free in quantities sufficient to overwhelm

bodily tissue on one occasion, and at another time may be so slight in amount as to cause but little if any disturbance. Again, toxins may vary in virulence from one day to the next. The mere occurrence of disturbances of the cardiac mechanism is by no means an indication that such disturbances are to be thenceforth permanently established; cardiac tissue can be transiently affected by toxins just as can other tissues of the body. He is a discreet electrocardiologist who refuses to commit himself to a prognosis on a patient who has been examined solely by graphic methods. The case should be studied clinically as well as cardiographically, and the graphic examination should be made under varying conditions of rest and exercise—thus recording many more cardiac events than does the usual single and isolated electrocardiogram which is taken solely when the patient is sitting and which covers in all, perhaps, only 40 cycles—40 seconds of the heart's day. Superficial observations and immature opinions, based upon the reading of a single and isolated electrocardiographic record, can bring nothing but disappointment to the physician and only discredit to the otherwise brilliant clinical future of electrocardiography.

CHAPTER XII.

The Significance of Blood-pressure Estimates.

PERHAPS it savors of injustice to describe sphygmomanometry as a procedure much practised and little understood. Such a statement, however, does the subject no more injustice than do ardent enthusiasts who assert that with elaborate blood-pressure formulas and a display of mathematics they can arrive at an accurate estimate of cardiac efficiency.

The pendulum of professional enthusiasm that swings too far to one side of the chronometer of facts, soon swings as far to the other. Between the extremes there is a center of balance where one may expect to find definite clinical values. Such definite values as the writer has found in blood-pressure estimates are expressed under "Blood-pressure Aids" farther on in this chapter.

Following the introduction, within the last decade, of several mechanical devices for estimating blood-pressure, the majority of the larger insurance companies of the United States have required that these clinical instruments be used by their medical examiners. The result has been a general adoption of sphygmomanometry, rarely with benefit to the applicant, frequently to the loss of otherwise acceptable risks by the insurance companies, and much to the confusion of the subject of sphygmomanometry. The inrush of physicians into a field where there had been

little opportunity to make the detailed studies required by the newer clinical method resulted in the publication of innumerable and hastily drawn conclusions; really competent observers have spent much time in disproving and controverting unfounded assertions, rather than devoting their attention to establishing sphygmomanometry upon a scientific basis.

IMPORTANCE OF COMPARATIVE READINGS.

For some inexplicable reason, sphygmomanometry is rarely practised in a systematic manner. It never occurs to many physicians, who take the temperature of a patient at each visit, to estimate the blood-pressure as frequently. It is in the *comparative* study of *frequent* blood-pressure estimates that its value to both patient and physician lies; cursory and perfunctory examinations are worse than useless in that they mislead. As an example of the value of repeated readings, it has been reported by Irving that, in the toxemia of pregnancy, a gradual and progressive rise in systolic pressure is of grave significance, and calls for energetic treatment.¹ When we have a similar frequency of observations, reported in a number of diseased conditions by a number of careful physicians, the subject of blood-pressure may be put upon a scientific basis.

THE APPARATUS.

There are two types of instruments—those which express the readings from a column of mercury within a graduated glass tube, and those operated by a spring. The former is considered more accurate.

¹ Irving: Jour. Am. Med. Assn., lxiv, No. 13, 935.

The latter is more convenient for clinical use, more easily carried and, if frequently corrected with the standard mercury scale, is sufficiently accurate for clinical use. It seems that the spring instrument is the one of choice with the majority of insurance companies who have answered circular letters on the subject; it is the one considered in this discussion.

The apparatus consists of a silk arm band five inches wide, which contains within its folds a rubber compression cuff. To one of the tubes supplying this air reservoir an atomizer bulb is applied; it has a provision for the gradual escape of air when desired. To the other tube the dial or register is attached.

The silk cuff is usually bound about the arm, over the biceps, in order to compress the brachial artery. The patient should be in the recumbent posture and the arm be parallel with the chest; inaccurate readings, both systolic and diastolic, result from either elevating or lowering the arm. There are many physicians who supplement observations made in the recumbent posture with observations made in the *standing* position, believing that by prolonging studies in the erect patient they can better estimate vasomotor instability.

The femoral artery may be used for blood-pressure estimates, but not with the patient sitting, for it then registers several millimeters higher than the brachial. In the recumbent posture the measurements taken at either point approximate each other. An exception to this statement is noted in cases of aortic insufficiency, in which condition the femoral reading in the recumbent posture may be 30 to 60 degrees higher than the brachial. The reading is also

higher if estimated through intervening sleeves; estimates made when the cuff is applied directly to the bare arm are more in keeping with the usual procedure.

TECHNIQUE.

Having applied the apparatus, place one hand at the radial artery, the other on the compression bulb. By compressing the bulb the pressure within the cuff is increased to a point a few degrees above that at which the radial pulse disappears. Gradually allow the air to escape; note that point at which the pulse reappears; this is called the *systolic* pressure. Carefully watching the dial as more air escapes, one will observe a point usually from 25 to 35 mm. below the high reading, where the greatest oscillation of the indicator takes place; this is called the *diastolic* pressure. When diastolic and systolic are subtracted, the resultant figure is called the *pulse-pressure*.

The above palpatory method should now be corroborated by the *auscultatory* method, suggested by Korotkow. This second reading, which is usually 5 to 10 mm. higher than that obtained by palpation, is accomplished by the simple maneuver of placing the bell of the stethoscope over the brachial artery, near the bend of the elbow, below the compressing cuff. Compress the bulb until the radial pulse disappears, then begin the gradual reduction of air. Note the point at which the sound first appears; a little further on this faint sound suddenly becomes snappy and rhythmical. This is regarded as indicating the systolic pressure. As the air in the cuff further declines there comes a point where the snappy and rhythmical

sound suddenly decreases in intensity. This is regarded as indicating the diastolic pressure.

The diastolic pressure is generally accredited with being an estimate of the degree of peripheral resistance, and an indication of vasomotor tone. Sewall,¹ however, believes it to be the measure of cardiac strain, and in support of this opinion points to the emptying of the heart-muscle vessels with each systole: during diastole the heart-muscle vessels are filled from the coronary arteries, "and it is a fair presumption that the volume of blood sent into the heart-muscle depends primarily on the diastolic arterial pressure, though the completeness of its removal is determined by the vigor of systolic contraction."

USUAL STANDARDS.

The estimates of Faught for arriving at the average standard of blood-pressure for a given age, are estimates much in favor. He gives the systolic pressure in a youth of 20 years as being 120: one degree is added for each additional 2 years of life; a standard variation of 17 in either direction is permitted. In women the reading is 10 mm. lower than in men.

A standard ratio is supposed to exist in health between the three pressures. It is called the "1-2-3 ratio," in which the diastolic pressure is twice the pulse-pressure; the systolic pressure is three times the pulse-pressure.

BLOOD-PRESSURE AIDS.

As concerns the *practical value* of sphygmomanometry as an aid in the appraisal of cardiovascular

¹ Sewall, Henry: Am. Jour. Med. Sci., vol. clviii, No. 6, p. 786.

conditions, the writer's experience has taught him as follows:

1. A systolic blood-pressure reading should be regarded as expressing in a figure scale some of the information which may be obtained by palpating an artery. It is an instrumental expression of tension; it reduces to the exactness of figures such terms as "full, bounding pulse;" "pulse of increased tension;" "low volume pulse" and other indefinite clinical phrases.

2. *Hypertension*, (hyperpiesis) or persistently high systolic readings, often direct our attention to systemic diseases which are reflected in cardiovascular derangements. Toxemia, nephritis, meningitis, exophthalmic goiter and syphilis are very frequently associated with high systolic readings.

Hypertension is also a frequent corroborative sign in such cardiovascular conditions as arteriosclerosis, aortic insufficiency, cardiac enlargement, aneurism and often in syphilitic aortitis.

3. *Hypotension*, or a lowered systolic pressure, is not of so much cardiovascular significance. It may exist in tuberculosis, in anemias, in Addison's disease, in hemorrhage and in shock.

4. *The exclusion value* of standard readings should not be lost sight of. Pressures constantly within accepted standards might aid in eliminating from consideration some of the diseased conditions mentioned above in paragraphs two and three.

BLOOD-PRESSURE "DON'TS."

1. Do not forget that blood-pressure is dependent upon (a) the force of the ventricular contraction;

(*b*) the degree of peripheral resistance; (*c*) the condition of the arterial wall; (*d*) the composition of the circulating blood—as well as upon other factors incident to the complex phenomena of life. It is therefore quite impossible to determine with a blood-pressure apparatus which of the many factors be at fault. So it is well not to commit oneself to a blind faith in suppositions drawn from blood-pressure readings alone.

2. Do not fail to remember that high pressure may be physiologic as well as pathologic. *It is not to be regarded as a disease.* Temporary rises observed in pain, neurasthenia, excitement and after exercise bear out the statement that pressure-rise may be physiologic. Even when persistently high in certain conditions, as may be the case in arteriosclerosis for example, it would seem to be Nature's method of supplying blood to vital tissues which otherwise might be more or less ischemic, owing to capillary fibrosis.

3. Do not imagine that elevated systolic pressure, alone considered, is diagnostic of cardiovascular disease. At best it can be only one of a group of many signs.

4. Do not assume that sphygmomanometric figures indicate the functional activity of the myocardium.

5. Do not expect clinical aid from the various mathematical formulæ whereby one is supposed to juggle blood-pressure readings into terms of heart-muscle efficiency.

6. Do not treat hypertension by administering circulatory sedatives—this may be meddling with Nature's safety-valve. Employ rational treatment, such

as rest in bed; freedom from anxiety, depressing emotions and excitement; free catharsis by the use of saline purges in small dose frequently repeated; elimination by the kidneys and by the skin,—these will reduce hypertension far more safely than will the employment of circulatory sedatives. Attention should of course be given to the diet, which should be carefully selected and of limited quantity. Foods that contain a high percentage of protein should be interdicted. Familiar examples of protein foods are meat, eggs, fish, shellfish, fowl, cheese, peas and beans. When emergency demands, venesection may be performed. Drugs should be employed only in exceptional instances, their power for good being questioned and their possibility for harm in other directions being generally admitted.

7. Do not overlook the therapeutic value of abdominal support, in persons with low systolic pressures and relaxed abdominal walls. Relaxation of vasomotor tone in vessels of the splanchnic area may be responsible for a sense of weakness, headache, dizziness, fatigue and other minor symptoms in a person of this type. A properly fitted abdominal belt frequently brings marked improvement in symptoms.

CHAPTER XIII.

The Irregular Pulse.

ABNORMALITIES of the cardiac mechanism are for the greater part reflected in the pulse. The Irregular Pulse has therefore been chosen as a chapter heading as being more expressive—even though it be a trifle less exact—than the preferred sentence Abnormalities of the Cardiac Mechanism.

GENERAL CONSIDERATIONS.

Certain pulse irregularities have no pathologic significance and are quite compatible with health,—as for example, sinus arrhythmia and occasional premature contractions. There are other pulse defects not yet advanced to a degree incompatible with health, which may foreshadow the onset of cardiac lesions and which require study at frequent intervals, examples of which are multiple premature contractions, actual dropped beats or other low-grade heart-blocks. Finally, there are pulse irregularities which are symptomatic of definite cardiac affections, as illustrated by the grossly irregular pulse of auricular fibrillation and by the pulsus alternans.

Abnormalities of the heart's mechanism may exist singly or in combination. They may or may not be accompanied by auscultatory phenomena and by valvular disease. Perhaps they are coincident with symptoms of circulatory failure—and perhaps they

are not. They may be acute—or they may be chronic. They may arise in the course of acute infectious processes or be attributable to other definite cause—or they may owe their inception to nothing that can be determined. With this general survey of attributes in common, let us now consider under individual heads abnormalities of the mechanism of the heart, bearing in mind that PULSE IRREGULARITIES ARE, FOR THE MOST PART, AN EXPRESSION OF MYOCARDIAL INVOLVEMENT. Their clinical significance lies in the fact that they are *an index of the degree of integrity of the heart muscle*—be the invasion of that integrity slight or serious, toxic or structural, transient or permanently established.

SINUS ARRHYTHMIA.

This condition, known also as the youthful type of irregularity, is characterized by a pulse that is irregular as to rate. It is clinically recognized by the fact that the pulse-rate increases on inspiration and decreases on expiration (Figs. 24 and 35). The irregularity disappears when the breath is held. It is of frequent occurrence in children and in the young, although not confined to those of tender years. It is observed occasionally in adult hearts, being more noticeable in emotional individuals. I noted an incidence of 12 per cent. in a group of neuro-circulatory asthenics that I studied. It may occur following exercise, when the heart has about recovered its rate from the enforced effort. There is evidence that excessive tobacco-smoking induces a fleeting period of sinus arrhythmia in hearts which

do not exhibit the phenomenon under moderate use of the leaf.

Sinus arrhythmia should be the first thought of the physician when a mother presents a child or a youth of school age with an irregular pulse; or the 5-second rate count (page 50) may first draw attention to it in routine examination. It is due to a change in vagal control and is not to be considered of pathologic significance.

The detection of this condition calls for no treatment. Neither does it indicate a curtailment of the usual activities of childhood. It usually disappears as the child matures. If sinus arrhythmia is accompanied by symptoms suggestive of circulatory disturbance, a search should be instituted for the complication which quite certainly co-exists.

PREMATURE CONTRACTIONS.

These are frequently miscalled *extra-systoles* and *dropped beats*. Both terms are misleading and grossly incorrect.

A premature contraction is a disturbance of the rhythm of the heart, wherein the heart contracts slightly in *advance* of the anticipated interval. Following this early contraction, there is a pause of longer duration than is customary in that pulse, after which *compensatory pause* the pulse again resumes its normal rhythm, until re-interrupted by a subsequent early beat.

As might be expected, a premature contraction is a weaker beat than its predecessor. The reason is clear: it occurs when the ventricle is not quite so full

of blood; it occurs before the ventricle has fully recovered its strength from the previous contraction; it occurs when the artery has not yet relaxed from the previous impact. So, it is a weaker beat. For the three directly opposed reasons, the *immediately succeeding* beat is stronger than other normal beats.

Premature contractions are believed to arise from an isolated and irritable focus in the auricle or in the ventricle; they may exceptionally arise from the atrio-ventricular node. The site of origin can be recognized only by graphic methods. Those arising in the ventricle are identified by the fact that the length of the period of disturbance plus the preceding beat is equal to the distance of two normal beats. In the auricular type these distances are *not* equal, nor does the jugular *a* wave in the polygram occur at the regular interval. Those of nodal origin can be detected only by electrocardiography. Ventricular premature contractions are found six times as often as are those of auricular origin.

One frequently observes isolated premature contractions in young persons whose previous history is irrelevant and whose subsequent cardiac course is entirely free from fault. Such a condition is likely to arise in persons whose duties are exacting and who have no opportunity to relax from continued physical effort; in some way the premature contractions seem to be associated with relaxed *tonicity* of heart muscle. Instances occur in physicians, for example, whose duties require that they be constantly on the move and in whom, after a winter of such nerve and physical strain, there may be noticed a disquieting irregularity in the action of the heart. A short vacation and

period of relaxation, followed by a reduction in the number of working hours and by the adoption of systematic exercises after the return home, usually cause such isolated premature contractions to disappear entirely from the pulse.

A premature contraction is the type of irregularity which patients seek to describe by saying that their heart "stops;" "turns over;" "drops a beat;" or else they affirm that their pulse is "intermittent." This, too, is what may be meant by the term "tobacco heart."

Questioning will develop the fact (when such symptoms are due to premature contractions), that the disturbance is *more frequent when the patient is at rest or relaxed*, and that it disappears upon exercise. This diagnostic point holds true whether the premature contractions are of the isolated variety or of the multiple-recurring type. *Increase in rate causes the irregularity to decrease or disappear.*

What is the significance of premature contractions? While any badly affected heart may show premature contractions, premature contractions do not mean a badly affected heart. Judgment would dictate that even if isolated and of rare occurrence in a patient who is otherwise symptom free, said patient might better be watched over a period of months until the harmless character of the irregularity is well established. Should they occur in hearts that did not show them previous to an acute infection which has put the patient in bed, it is the part of caution to regard premature contractions as evidence of myocardial damage of some degree. When they increase in frequency or when they are of the multiple- or

rapidly-recurring type (see Fig. 36) they may safely be regarded as quite indicative of heart damage.

The occurrence of premature contractions warrants a thorough cardiovascular examination, to ascertain whether or not they be associated with symptoms of circulatory fault. Treatment is directed to the cause underlying their production—whether it be systemic infections reflected in the heart's action, myocarditis, digestive or intestinal derangements in the aging body, or the result of nerve-strain, defective elimination, physical exhaustion, faulty habits or the habitual use of drugs.

PAROXYSMAL TACHYCARDIA.

Confronted by a patient who presents periods of rapid heart action, it is well to first establish which one of five possible conditions occasions the rapid heart. It may be caused by (1) ordinary tachycardia; (2) by hyperthyroidism; (3) by auricular flutter; (4) by auricular fibrillation; (5) by true paroxysmal tachycardia.

(1) *Ordinary tachycardia* is commonly the result of sympathetic irritation of the pacemaker; vagal depression may also be a cause. It occurs in overwrought individuals or in persons of the neurotic type. Loss of sleep, anxiety, nerve tension or emotion may be exciting factors. The rate reduces when the patient is at rest or asleep, or under the influence of nerve sedatives. The term "lability" is applied to the pulse which is ordinarily encountered in persons of an emotional nature. The word is derived from the Latin *labilis*, meaning "apt to slip." Hence a *labile*

pulse is one which exhibits sudden changes in rate due to emotional disturbances.

(2) *Hyperthyroidism* is likewise a sympathetic irritation, producing a tachycardia which is gradual in onset and gradual in decline. Clinical evidence of excessive thyroid activity such as enlargement of the gland, tremors and other nerve symptoms, eye signs, etc., point to the diagnosis and direct the treatment to the underlying cause of the tachycardia.

(3) *Auricular flutter* is often a 2 to 1 heart-block, giving rise to auricular rates between 200 and 350 per minute. The condition is separately discussed on page 144; it is mentioned here in order that it may be borne in mind as a form of tachycardia.

(4) *Auricular fibrillation* (discussed on page 145) may at times be ushered in by a period of very rapid heart action. One such instance which the writer has in mind gave a heart-rate of perhaps 220, with evidence of grave cardiac distress, dyspnea and cyanosis for two hours. Then general improvement and a lower rate followed, and the presence of fibrillation was announced by the grossly irregular pulse, disordered ventricular action and pulse-deficit characteristic of that condition.

(5) *True paroxysmal tachycardia* is a continued rapid succession of premature beats, recognized by the fact that the rate change is *absolutely abrupt* in onset and *absolutely abrupt* in termination. It arises from an abnormal irritable focus, which is usually located in the auricle (Fig. 37), rarely in the ventricle and which more rarely still may be of atrio-ventricular origin. Patients who have suffered from rheumatic fever or those who have arteriosclerosis are

the ones most frequently affected with true paroxysmal tachycardia; chronic thyroid patients are also liable to the paroxysms. It should be borne in mind however, that the condition may arise without presenting any suggestive cardiac history and not be at all accompanied by other evidence of cardiac disease.

The rate is rapid, usually from 160 to 180, best counted at the heart. This rapidity may persist for a few minutes or for weeks. Postural change does not alter the rate.

Prognosis is guarded but not grave. It is guarded because the attack may lead to exhaustion of the heart muscle, especially in the long-continued and frequently recurring cases. Short attacks, separated by considerable intervals of time, do not warrant gloomy pronouncements nor do they forecast a shortening of life.

Treatment during a paroxysm of tachycardia is directed towards conserving the heart effort by putting the patient at rest in bed. The patient will naturally assume that position which is the most comfortable to him; many prefer to be propped up on pillows. Diet should be light and easily assimilated. The organs of elimination should be kept active. An icebag applied to the precordium is helpful in some cases. Morphia may be required for insomnia. Supportive cardiac drugs are not as a rule indicated. There is some evidence that digitalis is effective in those rare instances where true paroxysmal tachycardia is of unquestionable *ventricular* origin. Pressure on the right vagus nerve, as it courses along the carotid sheath, is promptly effective in aborting many attacks. The pressure should be gently begun and

gradually increased to a point where the carotid pulsation is obliterated; carotid pulse obliteration is an indication that sufficient pressure is being exerted upon the vagus nerve.

Tachycardias of Undeterminable Origin.—One frequently encounters persistently rapid heart actions, no amount of study of which will permit their inclusion under one of the five sub-heads just considered. The tachycardia is of undeterminable origin. At a necropsy my attention was directed to a compression of the right vagus nerve, the structure being pressed upon by enlarged and calcified mediastinal glands. The possibility that such a circumstance might be one of the factors responsible for rapid heart rates of undeterminable origin caused me to enquire of anatomist friends whether or not such vagal compression was a rarity. Dr. Clarence Hoffman tells me he has repeatedly noticed in anatomic studies that the vagus has been pressed upon by enlarged mediastinal glands: Dr. W. F. R. Phillips has frequently observed such vagal compression, and was kind enough to furnish a photograph of a mediastinum which he was studying at the time of my inquiry (Fig. 47). Phillips is of the opinion that the right vagus nerve is more often compressed than is the left, for the reason that the right tracheo-bronchial chain of lymph glands is more numerous and the vagus more intimately in contact with this group than is the case on the left.

Whether or not enlarged glands, by long-continued pressure, could so compress the nerve as to interfere with its normal cardiac inhibitory function is a matter for conjecture. Such a diagnosis, as an ex-



FIG. 47.—VAGUS NERVE COMPRESSED BY LYMPH NODES.

The white arrow parallels the left vagus nerve. The black arrows point to enlarged and fibrous mediastinal glands which surround the nerve. (Dissection photograph through courtesy of *Prof. W. F. R. Phillips.*)

planation of a puzzling tachycardia must, from its very nature, remain purely hypothetical. However, the possibility is presented here for the satisfaction that it may afford the clinician in arriving at a tenable hypothesis concerning the cause of tachycardias which ensue in children or in adults who have had infections, such as tuberculosis, which involve acutely or chronically the tracheo-bronchial glands.

AURICULAR FLUTTER.

This is a condition in which an abnormal focus within the auricle gives rise to rapid, regular, pathologic impulses which cause the auricle to contract at rates varying between 200 and 350 per minute (Fig. 38).

This comparatively rare condition differs from true paroxysmal tachycardia in that the increased rate is almost invariably associated with a failure of conductivity. The ventricular rate is very often but *half* of the auricular rate, so that auricular flutter is really a 2 to 1 heart-block; not invariably so—but sufficiently often to warrant the statement. The electrocardiogram shows that the rapid auricle is driven at a regular and uniform rate of 200 or over.

In an elderly person who presents a persistent ventricular rate of over 120 per minute auricular flutter may be suspected. The suspicion is strengthened by the fact that the rate does not alter with change of position nor upon exercise. The ventricular rate may suddenly become half of what it was at the onset of the period of rapidity—a point which Lewis considers significant. Symptoms may be no more than those of

fatigue; profound constitutional symptoms, such as one would expect, are not usually present. The diagnosis can be absolutely determined only by an electrocardiogram.

Full doses of digitalis may be expected to change auricular flutter into auricular fibrillation, after which occurrence normal rhythm is resumed. I have also observed this same sequence of events in patients with auricular flutter to whom no drugs whatever were given.

AURICULAR FIBRILLATION.

HISTORICAL.

Auricular fibrillation has been variously known to physicians of past generations as "delirium cordis," "pulsus arrhythmicus" and the "pulsus irregularis perpetuus." Our present day knowledge of the condition has been but lately acquired, and is one of the debts we owe to polyography and electrocardiography—as indeed is our present knowledge of all pulse irregularities. Frank, in 1894 showed the effect of a fibrillating auricle on ventricular contraction. Cushing and Edwards contributed an article in 1906; but it remained for Lewis in 1909 to publish the first clinical paper on the subject, entitled "Auricular Fibrillation: a Common Clinical Condition."

DEFINITION.

Auricular fibrillation is a condition in which the normal and regular impulses for contraction, which arise from a *single* center, are replaced by a series of hap-hazard, *abnormal* impulses arising from a *number*

of irritable foci within the auricle. The auricle, instead of contracting at its usual rate in response to a normal stimulus, now stands in trembling diastole; for innumerable stimuli for its contraction are neutralizing their own effect in the musculature and virtually paralyzing the auricle.

The ventricle of course partakes of the confusion. *Some* of the abnormal stimuli from the auricle spread to the node of Tawara and result in ventricular contraction. Just as some of the concentric rings from an irregular succession of pebbles thrown into a brook will eventually reach the bank, so do some of the irregular impulses arising within the auricle reach the node which governs ventricular contraction. The result is haphazard ventricular action—now weak, now strong, never regular. An insufficient blood supply naturally results and congestion of tissues, visceral engorgements and the picture of circulatory failure ensue.

ETIOLOGY.

There are two causes of auricular delirium; toxins and diseased heart muscle. Toxins, for example, will cause perhaps 10 per cent. of pneumonia patients to fibrillate some time during the course of the lung condition. Many of the acute infections induce a temporary fibrillation; but the condition is far oftener *chronic* than temporary. In the infinitely more frequent chronic form the muscle of the auricle is found at necropsy to be either inflamed, degenerated or sclerosed. The valve lesion which frequently coexists with auricular fibrillation is mitral stenosis. Chorea and "rheumatic" infections—using the word "rheumatism" as the equivalent of a staphylococcic or

streptococcic infection—affect heart muscle and induce auricular fibrillation.

Arteriosclerosis is another frequent cause; the sclerosis involves the heart as well as the arteries. Fibrillation is often a terminal event in arteriosclerosis. It may rarely arise in aortic disease and exceptionally will be found in a patient of negative previous history.

CLINICAL RECOGNITION.

The Pulse.—In any pulse which is *continuously* and *persistently* and *absolutely irregular*, auricular fibrillation should be the first thought. This pulse varies in rate; it varies in rhythm; it varies in volume. Its count is not the same in two successive minutes; a pulse in the neighborhood of 120 one minute may be 100 the next minute. It may not at times be so fast—it may even be 60 or less, depending on whether the node of Tawara or the bundle be diseased. So while *rate* is not indicative, *irregularity of rate* is strongly suggestive of the condition. But most of all, *gross irregularity of rhythm and of volume* are essential to the diagnosis. On exceptionally rare occasions this pulse may be counterfeited by a marked sinus arrhythmia or by multiple premature contractions.

The Ventricle.—Associated with the *pulsus irregularis perpetuus* is tumultuous disorder of the ventricle, as revealed by auscultation at the heart. The ear is confused by a Babel of sounds; for an instant the physician believes he hears first and second heart sounds—then only the second is present; for a while a succession of peculiar little “runs” may be

discernible amid the cardiac tumult. Murmurs that were present before the inception of fibrillation either disappear or alter their character altogether; when the condition coexists with mitral stenosis, the characteristic presystolic murmur of the latter condition disappears. This murmur is produced by auricular contraction forcing blood through the narrowed mitral orifice: when the auricle is fibrillating it no longer effectually contracts—its action is virtually paralyzed; hence, no presystolic murmur is heard. An *early diastolic* murmur, due to ventricular filling, persists.

The Pulse Deficit.—Having noted first, the irregular pulse and second, the tumultuous heart, the examiner can now proceed to compare these two observations in their rates and develop the third sign of the diagnostic triad, *viz:* the pulse-deficit, or *the deficiency of the pulse as compared with the ventricular rate*. While one observer counts by auscultation the number of ventricular beats in a full minute, a fellow observer should count by palpation the radial beats *occurring in the same minute*, both using the same watch. The auscultator may announce a total of 120 beats; the one who is palpating the radial may have secured only eighty beats in the same minute. Subtracting the radial from the ventricular rate there results a pulse-deficit of 40 beats which is due to the fact that, in this particular instance, 40 of the attempted ventricular contractions were too weak to lift the aortic cusps and so failed to reach the wrist. It is little wonder that circulatory failure and threatened cardiac collapse so frequently attend what has well been called *delirium* of the heart.

Other Evidence.—The detection of this triad of signs—(1) the irregular pulse, (2) the disordered and often tumultuous ventricle and (3) the pulse-deficit—are sufficient grounds upon which to base a diagnosis of auricular fibrillation. Electrocardiography, the court of last resort, will quite invariably sustain the opinion established upon such evidence. Yet minor witnesses are not lacking with their corroborative testimony: fibrillators are notoriously of the “mitral type”—florid face, cyanotic lips, shallow respirations, anxious countenance and guarded physical movements all bear mute witness to a fibrillating auricle. A *history of embolism* may suggest the condition, for owing to the virtual paralysis of the auricle a clot may form in the auricular appendage (Figs. 2 and 3) and a detached particle be swept into the blood stream. Embolism is an unfortunate incident which sometimes occurs during treatment *just when the rhythm approaches normal*. Another symptom suggestive of fibrillation is *hoarseness*; this is occasioned by pressure made on the recurrent laryngeal nerve by the enlarged left auricle, which chamber may attain a surprising size, at times as great as the rest of the heart itself. As the patient responds to treatment the auricle lessens in size; the voice, if affected, improves.

The blood-pressure in auricular fibrillation, as the reader has already surmised, is always difficult—and frequently impossible—to estimate. The usual apparatus employed for the purpose gives no information of clinical value in this condition. Gaertner¹ devised

¹ Gaertner, G.: Ueber einen neuen Blutdruckmesser (Tonometer), Wien. klin. Wchnschr., 1899, xii, 696.

a method whereby a small pneumatic cuff is applied to the base of the ring finger, the finger rendered bloodless from tip to cuff, and the cuff attached to a mercury manometer. A certain set of figures were secured which are said to represent the blood-pressure in auricular fibrillation. It is not likely that one can obtain information of actual bedside value by employing the "capillary method" of Gaertner. As a usual thing, *auricular fibrillation is an evidence of serious damage to the musculature of the heart, and produces the signs and symptoms of circulatory failure: the indications for treatment are plain;* and such indications are in no wise made more absolute, nor modified, by capillary pressure estimates.

It might be well in passing to mention a sign of fibrillation which one is not always justified in eliciting. The irregularity of fibrillation *increases* when the sufferer is made to exercise; it decreases after a period of rest. On the contrary, other irregularities of the pulse *decrease* on exercise and re-appear on rest. It is often unwise to attempt to elicit this sign in a fibrillator who has much cardiac distress.

Electrocardiographic Recognition.—The curve of a normal heart, compared with a heart that is fibrillating, furnishes a comparison that requires no more explanation than will be found in the legend below Fig. 39.

PROGNOSIS.

Fibrillation, if acutely induced by toxins, may be of fleeting duration and never return. On the other hand, in wrecked hearts it may be the precursor of death within a few hours; or it may permit an

existence of cardiac invalidism for months. White found that 16 per cent. of a group of fibrillators died within ten months; from this one may deduce the gravity of the condition. To regard it properly, one should think of auricular fibrillation as an evidence of *damage* to the musculature of the heart. When, in spite of therapeutic efforts a ventricular rate of over 120 beats per minute is maintained, the outlook becomes progressively ominous in proportion to the rate maintained. The converse of this statement is also quite true.

TREATMENT.

Rest in bed is a therapeutic agent of no small value in auricular fibrillation. It slows the ventricular rate by lessening the systemic demand upon the cardiac muscle, thus often producing surprisingly beneficial results without resort to drugs. It is often good judgment, in the absence of urgent symptoms, to withhold drugs at first and deduce the value of absolute rest in bed by an estimate of the pulse-deficit at the expiration of 24 hours.

Digitalis is brilliantly effective in auricular fibrillation. Medical literature that was written long before auricular fibrillation was recognized as a clinical entity, describes typical cases of the malady and tells of the almost miraculous effects of the drug. Indeed, it would seem from testimony both new and old that upon the beneficent action of digitalis in auricular fibrillation rests the cardiac reputation of that drug.

Digitalis delays the conductivity of the atrio-ventricular node; in this manner it blocks off the shower of haphazard impulses that arise in the auricle.

Digitalis also steadies and increases the force of the ventricular contraction; in this manner are the symptoms of circulatory failure relieved. Bearing these facts in mind, its action in fibrillation can be readily understood.

Administration.—If a fibrillator is first seen with impending cardiac failure and *profoundly urgent symptoms*, a *physiologically tested* tincture of digitalis which has been *recently* tested may be administered *intravenously* in dosage equivalent to one and a half grains of the leaf. Unfortunately, the drug has not yet been *standardized*, in the usual acceptance of that term; a given quantity does not contain a definite amount of the active principle, for the reason that an active principle cannot be isolated (see “Cardiac Drugs”).

Desired results from the dose above mentioned may be expected within two hours. Should the physician have no information as to the strength of the tincture, it may be given in 30 minim doses at 3-hour intervals, by the mouth, *until results are obtained*, even though as much as 3 drams in 24 hours be thus administered. While this may, at first thought, seem to be a Herculean dose, it should be remembered that it is *emergency administration* and is intended for a fibrillator with impending circulatory failure and *profoundly urgent symptoms*. If the symptoms are less urgent the large dose mentioned above is not warranted, and 15 or 20 minims of a proved tincture may be given every 4 hours *until the heart rate approaches normal*. It is very convenient to watch the effect of drug administration by means of a chart, upon which is recorded observations made every 4 hours:

Patient _____	Date _____				
Hour _____					
Ventricular Rate _____					
Radial Rate _____					
Pulse Deficit _____					
Dose _____					
Preparation _____					

A ventricular rate brought to 85 per minute with a radial rate brought to 80 per minute may be considered a gratifying and desirable result in *the usual case*. In those usual cases which are met with in hospital wards, the heart rate may be expected to approach the individual's normal within three or four days. Digitalis may then be maintained in supportive dose of 8 to 10 minims of the tincture three times daily, to be gradually withdrawn and finally discontinued when there is no evidence of circulatory failure re-asserting itself after the patient is out of bed for several days. Following its withdrawal, the drug will maintain its effect on the heart for a variable period, lasting in some instances for three weeks. This continued effect of the drug might, on first thought, seem to explain the "cumulative action" of digitalis which is much referred to in text books of a decade past. It is probable, however, that "cumulative action" simply expresses the sudden inception

of heart-block, due to oversaturation of a digitalized atrio-ventricular bundle.

All fibrillating hearts will not react equally to digitalis; in some cases the heart muscle is but little damaged, in which event early improvement is the rule. In old and confirmed fibrillators the myocardium has undergone profound alteration and the drug may have to be continued comparatively long and in liberal dosage. No rule of dosage can be formulated to cover such diverging instances; administration of the remedy depends upon that universal rule of dosage, *viz*: —Administer at sufficiently frequent intervals in sufficient dosage to secure and maintain the results desired.

Digitalis should always be thoughtfully employed. It should not be given in trifling or inefficient dose; nor should it overwhelm a patient by long continued and unregulated dose. Coupling of the radial pulse beats is a signal for the cessation of digitalis. Its reckless use may induce a profound cardiac emergency.

Morphine is a dependable drug in fibrillation. It slows the heart rate, not by direct action on the cardiac muscle for there it has no effect, but by stimulation of the vagal center. It relaxes nerve tension; it induces sleep. Myocardial damage does not contraindicate morphine except in so far as its effect in large or long continued dose may, by its effect on remote organs, add to the heart's embarrassment. The sulphate may be administered in dosage of $\frac{1}{4}$ grain, to be repeated in 2 or 3 hours if the desired effect is not secured.

Strophanthus.—*Digitalis* may occasionally induce gastric symptoms and not be well tolerated by a patient. Therefore its fellow-drug, *strophanthus*, is often used as a substitute. But recent observations indicate that *strophanthus* may overwhelm a patient who has *already received digitalis* and sudden death result. *Strophanthin* has proved markedly effective when employed intravenously as the sole medication in auricular fibrillation.

Venesection may prove to be a life-saving procedure in desperately urgent cases of auricular fibrillation, where to wait for the action of drugs is an unwarranted delay. It relieves the embarrassment of the distended auricle, thus lessening the load of the heart. As much as 16 or 20 ounces of blood may be withdrawn from an opening made in the median basilic or other convenient vein, always under aseptic precautions.

SUMMARY.

(1) Auricular fibrillation is the most frequent factor in the production of an irregular pulse which is associated with symptoms of circulatory failure.

(2) It is readily diagnosed clinically by the fibrillation triad, *viz*; irregular pulse, abnormal ventricular action and pulse-deficit.

(3) Exceptional or complicated cases may require electrocardiographic examination to establish the diagnosis.

(4) The prognosis is always serious and often grave.

(5) *Digitalis* is brilliantly effective in this condition, but is to be employed with judgment.

HEART-BLOCK.

Heart-block is a term that defines the blocking of the impulse for contraction at some point in its course from the pacemaker to the final distribution of the fibers of Purkinje.

LOCATION.

Block may be located at any point along the conduction system. If it be situated at the junction of the pacemaker with the auricular muscle it is called *sino-auricular block*. It may occur in the *A-V* node, in the bundle of His, in the right or left branches of the bundle—in which latter situation it is called *bundle branch block*—and even in the Purkinje fibers, where it is called *arborization block*. (Fig. 43.)

VARIETIES OF BLOCK.

(1) Block may be “*functional*,” as in the sudden halving of rate which sometimes occurs under digitalis administration. (2) *Transient* block may arise during the course of disease, as in rheumatic fever or erysipelas. A patient with the latter condition whom the writer studied would, during his convalescence, alarm his nurse by a symptom-free drop in pulse-rate from 80 to 35; after a few hours, normal rhythm would be resumed. He repeated this performance at intervals over a period of four days as he lay comfortably in bed—but emerged from the hospital apparently none the worse for his pulse vagaries. (3) *Organic* block is illustrated by the sclerosed bundle of His sometimes found at necropsy in syphilitic hearts.

Block is also referred to by *grades*. *Low grade* blocks are those in which many of the impulses originated by the auricular pacemaker result in ventricular contraction (Fig. 41). *High grade* blocks, which occur for the most part in males over 50 years of age, are illustrated by bundle branch blocks and by complete heart-block (Figs. 27 and 42). In the latter condition there is complete dissociation of auricles and ventricles; the auricle contracts regularly in response to its usual impulse, but the impulses are blocked and do not come through. The ventricle then initiates a rhythm of its own (usually in the neighborhood of 30 beats a minute) and its contractions are absolutely independent of any auricular impulse.

DEGREES OF BLOCK.

There are degrees of block, varying progressively in severity, as enumerated below:

- (1) Delayed conduction (*P-R* or *a-c* interval over 0.2 of a second). (Fig 40.)
- (2) Actual dropped beat (Figs. 26 and 41).
- (3) 2 to 1 block (2 auricular to 1 ventricular contraction).
- (4) 3 to 1 block—etc.
- (5) Bundle branch block.
- (6) Complete dissociation (Figs. 27 and 42).

CAUSES OF BLOCK.

The loose administration of digitalis is responsible for many of the heart-blocks that enter hospital wards. Rheumatic fever and diphtheria are productive of heart-block. Arteriosclerosis and block frequently co-exist. Syphilis notoriously invades the in-

tegrity of the conduction system. Lesions of the aortic valve, often of specific origin, may extend to the bundle of His, which divides under the aortic cusps. Whatever the provocative disease may be, these observations lead us to a fundamental conception underlying the production of heart-block, *viz*; *heart-block should be regarded as but part of a more widely spread involvement of cardiac tissue*. It is not at all probable that disease confines its ravages to the conduction system alone; it is far more reasonable to suppose that the slender conduction system is involved together with other cardiac tissue. This hypothesis is sustained by clinical observations and from necropsy findings. Patients do not die from any condition peculiar to heart-block; they die with the usual symptoms of heart failure.

DIAGNOSIS.

Suspicion of heart-block should be aroused when there is a slow pulse-rate of fifty or less. Suggestive, too, are 4 or 5 waves in the jugular to one pulsation at the ventricle—but remember that a possible *h* wave in the jugular robs this sign of its pathognomonic significance. Gallop rhythm, reduplication of the heart sounds and actual dropped beats at the wrist are significant. But the latter, to be genuine, must be coincident with a period of silence at the ventricle. Care should also be taken not to confuse a dropped beat with a premature contraction that happens to be too weak to lift the aortic cusps; such a contraction is recognized by having a first sound but is minus the second sound. The *Stokes-Adams syndrome*, described by Adams in 1827 and elaborated

upon by Stokes in 1836, occurs in heart-block when ventricular silence lasts over 3 or 4 seconds. It is caused by an arrest of blood-supply to the brain and is characterized by slow ventricular rate, syncope and epileptiform convulsions. Milder degrees of this syndrome are far-away sensations, dizziness and a momentary loss of consciousness with perhaps muscular twitching.

The writer has observed in the higher grades of heart-block that auricular activity, while impossible to detect through the stethoscope, can frequently be appreciated when the ear is placed directly over the auricle.

Graphic records furnish proof positive of heart-block. The electrocardiogram will refine the diagnosis to the point of telling whether the right or the left branch of the bundle be affected; even more, it will indicate lesions of the fibers of Purkinje in either the right or the left ventricle. (Fig. 43.)

PROGNOSIS.

The prognosis in heart-block is to be based on the degree of heart muscle involvement. Cardiac disease that has progressed sufficiently to seriously affect the conduction system, as in either bundle-branch block or complete heart-block, rarely offers a life expectancy of over three years.

TREATMENT.

Patients with the lower grades of heart-block need not be confined to bed, but may be up and around. Patients with the higher grades may attend to some of their accustomed affairs, unless signs of cardiac

failure supervene. The treatment of heart-block is directed to the underlying condition; if syphilis be the causative factor, for example, antisypilitics may arrest the progress of the lesion. Epinephrin, in solution equivalent to $\frac{1}{65}$ of a grain, has been used subcutaneously with prompt effect in toxic block. Atropine, in $\frac{1}{100}$ grain doses, may result in the re-establishment of normal rhythm in blocks of milder degree. If digitalis be employed to support a failing heart, it must be cautiously employed, for *digitalis may convert an incomplete block into a complete block*. Of course, if complete block already exists, digitalis cannot make it any more complete and, by its action on cardiac muscle, may prolong life. The only contraindication to digitalis in complete block is the presence of the Stokes-Adams syndrome; its use under such circumstances has proved perilous.

PULSUS ALTERNANS.

This is a condition in which every other pulse wave varies in height. The pulse alternates in volume. It alternates because certain muscle-fibers of the heart are too exhausted to respond to each and every impulse for contraction. Hence the physiologic function of *contractility* is interfered with, and it is a sign of grave heart damage.

Alternation is often observed in patients who have arteriosclerosis or angina pectoris—both of which conditions, if of specific origin, might be regarded as but local expressions or a more generalized cardiovascular involvement that has invaded heart muscle as well as arteries. Likewise, alternation may be seen

occasionally in hearts exhausted from other causes, as in acute infectious diseases. In short, *alternation is associated with conditions which produce exhaustion of heart muscle*. When it arises transiently in the young, as an incidence of acute infection, it is of course not of the same sinister significance as when it is permanently established in the aged.

DIAGNOSIS.

The smaller alternating beat may be so little smaller than its predecessor that one misses the diagnosis. It may require gradually increasing pressure on the brachial artery to obliterate the weaker beat to a point where the radial rate is cut in half. Frequently the condition is first discovered while estimating blood-pressure, (when the arm band is inflated), and is announced by a sudden halving of radial rate. The polygram faithfully records pulsus alternans. It is well to habitually look for alternation following ventricular premature contractions; they often initiate a period of alternation (Fig. 28). Remember that alternation must alternate. It must occur every other beat; if it occurs every third or fourth beat it is not alternation. As a general proposition, the more continuous the runs of alternation in the tracing, the more serious the involvement; the lesser the degree of amplitude in the smaller waves, the graver the prognosis.

The detection of pulsus alternans calls for all measures that will conserve heart effort. These measures may be tersely summed up as rest, elimination, dietetic regulation and, if required, supportive cardiac drugs.

There are other abnormalities of the cardiac mechanism, in addition to the seven conditions considered in this chapter. They include such phenomena as atrioventricular rhythm, auricular standstill, ventricular escape, ventricular flutter, etc. Inasmuch as these are recognized only by electrocardiographic studies, their inclusion here would be beyond the intent of a clinical manual.

THE SIGNIFICANCE OF CARDIAC IRREGULARITIES.

It is important, for clinical purposes, that the physician grasps the true significance of disturbances of the cardiac mechanism—whether or not they produce an irregular pulse by which they can be recognized at the bedside or whether they are detected only by cardiographic methods. That true significance is this: in the first place, disturbances of the cardiac mechanism are usually the result of a disturbance of the conduction system (Fig. 7). The disturbance may be transitory, or it may be permanently established. It is highly probable that affections of the conduction system are simply an evidence that the conduction system has shared in a pathologic process that has involved the conduction fibers as well as the heart muscle. Neither logic nor analysis will permit one to assume that the slender, delicate conduction system alone can be invaded by disease while the muscle tissue, in which these fibers are so intimately distributed, goes scot-free from similar invasion. Therefore, disturbances of the heart's mechanism can safely be regarded as *an indication of the degree of integrity of the heart muscle*. If this be true, it behooves the

physician to closely observe patients so affected, in order to ascertain whether the cardiac disturbance be progressive in character—and, if progressive, to institute treatment in the hope of anticipating and preventing the eventual *heart failure* which pulse irregularities so often foreshadow.

CHAPTER XIV.

Pericarditis.

PHYSIOLOGIC FUNCTION OF THE PERICARDIUM.

THE pericardium in health is regarded as an inextensible membrane which supports the heart and is capable of resisting an intense pressure. This view of the pericardium applies only in health; for its fibers are relaxed and stretched to a much greater capacity when the sac is inflamed or when a gradually accumulating effusion produces a sustained pressure within the sac. Recent experiments have confirmed the restraining influence of the pericardium upon the heart. Experimental removal of the pericardium in animals has shown that it is necessary for the normal mechanism of the heart. In the animals subjected to this experiment there ensued irregularity of the heart beat, venous overdistension and incompetence of the mitral and of the tricuspid valves, with eventual rupture of the left ventricular wall.

CLASSIFICATION OF PERICARDITIS.

Inflammation of the pericardium may be clinically classified as (1) *Acute pericarditis* (also called dry, plastic, villous or fibrinous); (2) *Pericarditis with effusion* (either serous or purulent); (3) *Pericarditis with adhesions* (adherent pericardium). These conditions may be progressive steps from one to the other,



FIG. 48.—HEMOPERICARDIUM.

The heart is encircled by a mass of clotted blood which has ruptured from the aorta into the pericardial sac. (Jefferson Medical College Museum.)

—although neither condition necessarily develops into its successor; the inflammation may subside at any stage. Rarer involvements of the membrane are (4) *Hydropericardium*, which is a transudate occurring as a part of a general anasarca; (5) *Hemopericardium*, where blood escapes into the sac as a result of injury or accident; (6) *Pneumopericardium*, due to air or gas in the sac. The last three mentioned conditions are of such unusual occurrence and diagnosed so infrequently that they may be conveniently disposed of at this point.

Hydropericardium rarely manifests itself by clinical signs, for it is non-inflammatory in character and the fluid is of moderate quantity.

Hemopericardium is a rapidly fatal accident which has been found at necropsies to have resulted from such conditions as the rupture of an aneurism in that part of the aorta which is situated within the pericardial sac (Fig. 48), or from the bursting of a coronary artery, or from rupture of the heart muscle. It may also arise as a result of puncture wounds of the pericardium. Whatever be its accidental cause, death usually takes place before clinical symptoms or physical signs sufficient to recognize the condition have manifested themselves.

Pneumopericardium is generally associated with an effusion, the decomposition of bacteria therein giving rise to the gas that is in the sac; or it may arise through a fistula which opens into the pericardium from an adjacent organ. In less urgent cases of pneumopericardium the symptoms are those of pericarditis; percussion may establish tympany over the upper area of the sac; below there is a flat note,

due to the presence of liquid. Auscultation may reveal a metallic gurgle which is synchronous with the movements of the heart. The condition is not to be confused with air in the *pleural* cavity, nor with a stomach which is distended with gas.

It is, however, with the first three mentioned varieties of pericarditis that the physician will have his principal clinical dealings, and they will therefore now be considered at greater length.

ETIOLOGY OF ACUTE PERICARDITIS.

Acute pericarditis is rare as a primary event. It is usually secondary to infection elsewhere in the body. The statistics of Robey¹ indicate that out of 78 cases of acute pericarditis which came to autopsy, 58 of them, or 74.4 per cent., presented some form of acute infection before death; acute pneumonia or pleuritis was found 49 times, or in approximately 63 per cent. of the cases. Acute pericarditis occurred in scarlet fever 5 times, (6 per cent.), and in diphtheria 4 times, (5 per cent.); in chronic interstitial nephritis 3 times, (4 per cent.); in cardiac disease 4 times; in malignant endocarditis 3 times, etc.

Strangely enough, the clinical diagnoses in Robey's statistics do not include acute arthritis, despite the general clinical observation that pericarditis is more apt to manifest itself in acute arthritis than in any other primary infection. In the statistics reported by Garrod² pericarditis was observed 344 times in 2,080 cases, (17.1 per cent.), of acute articular rheumatism. It is held by many physicians that the more

¹ Robey, W. H. Jr.: Am. Jour. Med. Sci., Apr., 1917.

² Garrod, A. E.: "A Treatise on Rheumatism."

severe the arthritic involvement, the more likelihood there is of pericarditis occurring. It is not uncommon for acute pericarditis to occur on the third or fourth day of an arthritis and Balfour thought that the majority of the cases occurred within the first week. The frequent incidence of the disease in childhood suggests that the pericardium should be carefully watched in the "rheumatic" affections of children. Tonsillitis and chorea may occasion pericarditis. Tuberculous involvement of the pericardium is of infrequent occurrence. Extension of inflammation from nearby structures is a possibility, although the great majority of pericardial inflammations are secondary to infections carried to the membrane by the blood stream.

DIAGNOSIS OF ACUTE PERICARDITIS.

Acute pericarditis is a condition which is frequently overlooked. The history of 34,647 patients at one hospital shows that it was clinically recognized in that number of cases only one hundred times. The disease so often fails to announce itself by physical signs that its discovery is often accidental, its symptoms being overshadowed by the severity of the primary infectious process to which the pericarditis is secondary. The diagnosis rests principally upon the detection of one physical sign, *viz.*, "*The to-and-fro friction rub*," which can sometimes be felt as well as heard. Pericardial friction is occasioned by the rubbing together of the parietal and visceral layers of the pericardial membrane, the surfaces of which are roughened by inflammatory exudates (Fig. 49). Manifestly if the inflammation be slight or the ex-



FIG. 49.—ACUTE (VILLOUS) PERICARDITIS.
(University of Pennsylvania Medical School Museum.)

update be small in amount, the sound produced is less intense and more resembles the rubbing together of parchment surfaces, rather than the heavy, coarse sound which occurs in profound inflammation with thick exudate, to which the classical term "leathery friction rub" is applied. As a matter of fact, the sound does not always reach the intensity of that produced by the rubbing together of leather surfaces.

Pericardial friction is interrupted in character, the interruptions being quite similar to those produced by rubbing the balls of the thumbs over each other. It has a varying relation to the sounds of the cardiac cycle, and gives the impression of being more superficial in character than the sounds produced by the valves of the heart. Pericardial friction, while it may be heard at various points over the heart, is best heard to the left of the sternal border at the junction of the 3d or 4th rib, for it is in this area that the pericardial surfaces come in closest apposition to the wall. It is clear that if one listens for the friction rub at this area when the patient is erect, he may intensify the sound by moderate pressure with the stethoscope; it is equally clear that heavy pressure at this area may utterly obliterate the sound, inasmuch as it may force the pericardial surfaces together. The sound, which is best heard when the patient is in the erect or sitting posture, becomes less audible when the patient is recumbent; should the pericardial inflammation progress and effusion take place, the gradual accumulation of fluid may separate the pericardial layers so that they no longer touch each other when the heart moves, and the sound will be no longer heard. The disappearance of the friction rub, then,

may mean either that (1) the exudate is subsiding, in which event we would expect an improvement in the general condition of the patient, or that (2) effusion is rapidly forming, a circumstance which we would expect percussion to confirm. It must be borne in mind, however, that the sound of pericardial friction may vary in intensity and in location from the time of one examination to another, or may be made to vary with changes in posture of the patient.

Pain is not a striking symptom in pericarditis; rather, its absence is striking. There may be a sense of pericardial distress, but the patient does not actively complain of pain in the majority of instances. Mackenzie believes that pain is present only when the myocardium is involved. Fever is an inconstant symptom and, when the temperature is elevated as a result of the pericardial involvement alone, amounts to only a slight rise.

Pulmonary compression signs in acute pericarditis have recently been described by Christian.¹ They are not to be confused with the symptoms of pulmonary compression which occur in pericarditis with *effusion*, to which attention was drawn years ago by Ewart. Christian states that of 33 patients in whom the diagnosis of acute pericarditis was made because of an audible friction rub 39, or 73 per cent., showed pathologic changes in the left lower back. These changes consisted of dullness, usually distinct, which occupied a small area at the angle of the scapula. More extensive processes occupied the lower third or half of the back. Over the dull area

¹ Christian, Henry A.: Jour. Am. Med. Assn., vol. lxxi, No. 6, p. 419.

bronchial breathing of varying intensity and bronchophony could be heard. In a few instances the detection of these phenomena enabled the writer to make the diagnosis of pericarditis some days prior to the advent of the typical pericardial friction rub.

The breathing rate in acute pericarditis is usually increased, sometimes markedly so. An example of this was seen in an American soldier who was brought from a battle field in France to an evacuation hospital near the front, fifteen hours after being rendered *hors de combat*. He was perfectly conscious; had slight precordial discomfort, and a pulse rate of 120, with respirations which averaged 40 per minute when at rest. Fever was absent. There was no evidence of respiratory infection. His leg had been shattered by the bursting of shrapnel, and at that time he noticed that his breathing became short and rapid and remained so. X-ray examination of the chest revealed a piece of shrapnel, approximately the size of a lead pencil eraser, which had penetrated the pericardium and which was held in position there. The patient died a few hours afterwards, before he could be brought to the operating table. The post-mortem records did not show any pathologic condition other than the foreign body which could have been responsible for the increased respiratory rate, thus permitting the deduction that irritation of the pericardium had produced the hurried breathing.

DIAGNOSIS OF PERICARDITIS WITH EFFUSION.

Experimentally the average pericardial sac will hold approximately 1½ pints without over-distension. It is reported in medical literature that as much as a

gallon of fluid has been taken from a patient suffering with tuberculous pericarditis, and that the patient subsequently recovered. Another patient with tuberculous pericarditis is known to have borne a quart of fluid in the pericardium with surprisingly little discomfort. The amount of fluid, it is thus seen, may vary from a slight excess of the two ounces or less



FIG. 50.—PERICARDITIS WITH EFFUSION.
Showing the heart within an enlarged and thickened pericardial sac.
(University of Pennsylvania Medical School Museum.)

(50 c.c.) which is normally in the sac, to the surprising quantity of $8\frac{1}{2}$ pints (4000 c.c.). (Fig. 50.) Cabot states that less than 5 ounces is not clinically demonstrable.

It is therefore seen that the physical signs of pericardial effusion will vary in proportion to the amount of fluid which has accumulated at the time of a given examination; the symptoms too, will vary, according to whether the fluid be serous or purulent, and accord-

ing to the degree of mechanical pressure which the accumulation of fluid exerts upon either the heart itself or upon adjacent structures.

Physical Signs.—In the young child *inspection* may reveal bulging of the left thoracic wall with obliteration of the interspaces on that side. The more resisting adult chest does not admit of such changes taking place. *Palpation* may confirm the impression of fullness as above noted in children; it may also detect the pressure of a pericardial friction rub if one be present, and by palpation one may further note the absence of the cardiac impulse, should sufficient fluid be interposed between the heart and chest wall. *Percussion* is of value in determining the presence of pericardial effusion by increase in the usual area of cardiac dullness; it further outlines the extent of the accumulated fluid. The usual area of cardiac dullness is found to be considerably increased to the left, and if the amount of fluid be sufficient, dullness will extend upward over the heart, giving the classical “pearshaped” area of cardiac dullness, in which the stem of the pear is directed upward. Extensive effusions may also obtund Ebstein’s cardio-hepatic angle, which angle is formed by the close apposition of the right border of the heart with the liver (see Fig. 51). Changing the position of the patient may cause the limits of dullness to change their position, as the fluid is shifted from one side to another. The same maneuvers may be responsible for the appearance and disappearance of the cardiac impulse, due to the interposition or recession of fluid. The *x-ray* may be requisitioned to confirm the information above elicited by percussion (Fig. 52).

Auscultation is of value in that it shows a gradual decrease in the intensity of the normal heart sounds from day to day as increasing fluid interposes itself between the heart and stethoscope. It may also note, as the fluid is absorbed, increasing strength of the normal sounds which previously had been feeble, muffled, or perhaps altogether absent. Auscultation

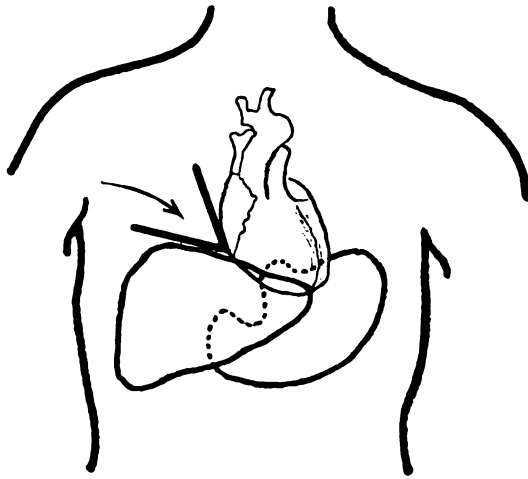



FIG. 51.—THE CARDIOHEPATIC ANGLE.

frequently detects, as has already been pointed out, the gradual disappearance of the pericardial friction rub as fluid accumulates, or may note its reappearance from time to time as the fluid alters in position.

Pulmonary Signs.—Auscultation of the left lung posteriorly, particularly at the angle of the scapula, very often reveals patches of dullness which are believed to be due to compression of the lung by the pericardial effusion. Over such patches of dullness the vocal fremitus is greater than is that of the opposite side, and tubular breathing may also be heard.

Effusion in the pericardial sac may be of such degree as to extend to the left mid-axillary line producing an area of dullness in this region, at the upper margin of which Skodaic resonance frequently exists; the breath sounds and the voice tones are altered. This latter group of signs may be made to alter in character or shift in position by making changes in the posture of the patient. It is well to bear in mind that the pulmonary signs which occur in pericarditis are also found in a slowly clearing basal pneumonia or pleural effusion.

Differential Diagnosis of Pericardial Effusions.—When differentiating *pericardial effusion* from a left-sided *pleural effusion*, one will recall that in pleurisy there is a history of sharp pain in the side; that a *pleural* friction rub bears no relation to the events of the cardiac cycle, and can be made to disappear when the patient is instructed to hold his breath; and that furthermore, the upper limit of dullness in pleural effusion often takes the shape of the letter “S,” (turned on its side, as ) should it extend from the anterior to the posterior chest wall. In pleural effusion there is not the diminution in the normal sounds of the heart which are to be expected in pericardial effusion, nor does the percussion note change so abruptly from dullness to resonance as when outlining the intrapericardial accumulation. To differentiate *pericardial effusion* from *cardiac enlargement*, it is of assistance to remember, after one has considered the difference in histories, that in cardiac enlargement the maximum cardiac impulse can usually be located without much difficulty and that the outer limit of the maximum cardiac impulse well defines the

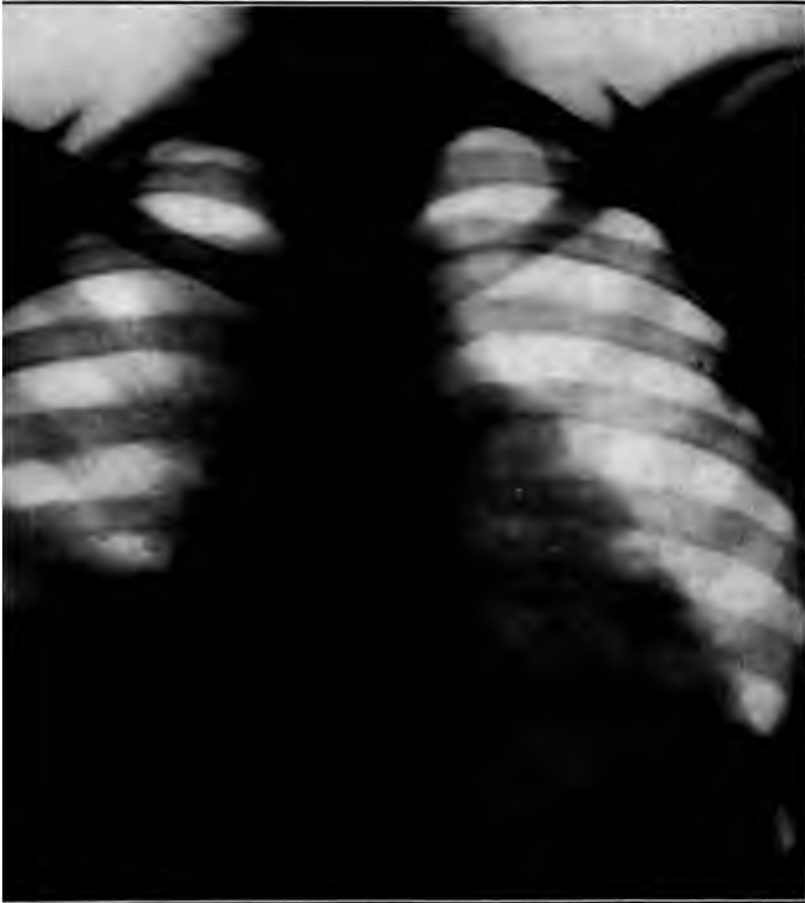


FIG. 52.—PROBABLE PERICARDIAL EFFUSION.
(Courtesy of *Dr. Willis F. Manges.*)

point at which cardiac dullness disappears. In border line cases the fluoroscope or skiagraph may establish the diagnosis.

Exploratory puncture of the pericardium has little justification. To introduce a hollow needle for the sole purpose of determining whether or not fluid be present is a confession of weakness in the art of diagnosis. On the other hand, to introduce a needle for the purpose of differentiating between a *serous* and a *purulent* effusion may be a justifiable procedure. Purulent effusions are frequently of such small quantity that one fails to attribute to their presence the prostration, repeated chills, septic temperature, leukocytosis and emaciation of a patient; especially is this so if a previously large effusion has decreased in size through partial absorption of its more liquid constituents, permitting pus of a creamy consistency to remain. Under such circumstances one is warranted in employing the hypodermic needle for diagnosis. The pericardium may be entered at the sites mentioned under "Paracentesis" in the paragraph on treatment which follows.

DIAGNOSIS OF PERICARDITIS WITH ADHESIONS.

Pericardial inflammation frequently terminates in the formation of adhesions. When these adhesions form between the parietal and the visceral pericardium they are known as (1) *Intrapericardial* adhesions. When they form between the parietal pericardium and adjacent structures they are (2) *Extrapericardial* adhesions, and the condition is then called *mediastino-pericarditis*. It will sometimes be found

at necropsy that the visceral and the parietal pericardium are bound closely together, forming a dense and inseparable structure, and only to such a condition is the term *adherent pericardium* properly applied.

(1) *Intrapericardial* adhesions have a tendency to dispose themselves over those areas of the heart where the visceral and pericardial layers come in closest apposition to each other. For this reason adhesions will be found near the apex of the heart, in which situation they may be rather dense and fibrous owing to the tug exerted upon them by the apex. They also form along the outer border of the left ventricle and over the left auricle, and at the base of the great vessels. A network of adhesions may form around the left auricle or around the ventricle or at the base of the great vessels, enmeshing these parts; or the adhesions may be slender and not sufficiently extensive to be of any clinical significance.

(2) *Extrapericardial adhesions* may be attached to the lungs, to the diaphragm, to the sternum, to the ribs and even to the vertebræ. Such adhesions are believed to be the result of distension of the pericardial sac with fluid which has accumulated during the progress of a severe pericardial inflammation, at which time the attachments took place (Fig. 53). Statistics of 80 cases of mediastino-pericarditis revealed the fact that 49 of the hearts thus affected had also co-existent valvular lesions. Whether the valvular lesions under such circumstances are the result of a probable pancarditis which damaged all the cardiac structures at the same time, or whether the valvular lesions resulted from the extracardial adhesions which deprived

the heart of the support normally afforded by its non-elastic membrane, is a debatable point which the clinical history of a given case might decide.

Physical Signs.—Intrapericardial adhesions do not yield any definite symptoms or signs which would lead to their recognition. Mediastino-pericarditis, on the other hand, affords symptoms and signs which depend for their severity upon the extent of the attachments between the pericardium and other structures. Significant symptoms are found in a history suggestive of previous pericardial inflammation; in irregularity of the heart, in dyspnea upon slight exertion, in precordial distress or in moderate *anasarca*. A physical sign of importance is a limitation in movement of the apex: in a normal heart or even in an enlarged heart the apex will move one and one half or two inches to the left when the patient is placed upon the left side. In mediastino-pericarditis the apex will not shift but will remain stationary during this maneuver. Some significance might perhaps be attached to the character of the maximum cardiac impulse which is often strong, sharp and quick. Should the adhesions be formed between the *heart and lungs*, it is sometimes possible to notice the maximum cardiac impulse ascending when the patient's left arm is raised over his head. Adhesions that form *posteriorly* will give us the sign described by Broadbent, in which there is systolic retraction of the 10th and 11th interspaces, visible in back. When the pericardium is adherent to the *diaphragm* there is a limit to the respiratory motion, which reduction in motion can be appreciated by the eye or felt with the hand when compared with the respiratory movement of the other



FIG. 53.—PERICARDIAL ADHESIVE BANDS.
(University of Pennsylvania Medical School Museum.)

side. Adhesions to the *sternum* may show that the normal respiratory increase, amounting to an inch in the anterior-posterior diameter of the chest, is limited in extent. Calipers may be used to determine the extent of such limitation.

The term *pulsus paradoxus* defines a pulse which varies in volume with the respirations of the patient. It becomes larger and stronger during expiration; this is exactly opposite to a normal pulse, and hence the term *paradoxical* is employed to define it. For many years it has been taught that the *pulsus paradoxus* is symptomatic of pericarditis with adhesions. It is no longer considered of diagnostic significance in pericarditis, for it occurs in conditions which are associated with heart muscle damage and in which pericardial adhesions have not been found.

Despite the above mentioned diagnostic clues, there are many cases of adhesive pericarditis which afford no evidence whatever of their existence and are discovered only at necropsy. Especially is this so when the condition co-exists with valvular defects, for to the leaking valve is credited the occurrence of symptoms and signs which might otherwise be attributed to pericardial fault. Again, the cardiac disturbances incident to a valvular defect may overshadow any evidence of pericardial inflammation.

DIAGNOSIS OF ADHERENT PERICARDIUM.

Adherent pericardium does not afford any definite symptoms or signs by which it can be clinically recognized (Fig. 54). Its presence is often suspected if there be marked enlargement of the heart which cannot be attributed to valvular defects, to affections of

the vessels or to cardio-renal disease. If in addition to this there be a history of rheumatic fever or of other acute inflammation, with the history of acute



FIG. 54.—ADHERENT PERICARDIUM.

The pericardial sac is intimately adherent to the heart. (University of Pennsylvania Medical School Museum, collection of Dr. R. S. Willson.)

pericarditis or endocarditis at that time the suspicion becomes of more value.

Kussmaul drew attention to *systolic filling of the cervical veins* as a suggestive sign upon inspection. Broadbent described a *diastolic shock* due to aortic

closure, which, however, also occurs in hypertension and in aortic dilatation. The pulse exhibits nothing of any significance, and may take on the character of a co-existing valvular lesion; for example, if there be an associated aortic insufficiency, the pulse may be of the quickly collapsing character described by Dominic Corrigan. Auscultation affords nothing distinctive by which adhesive pericarditis can be recognized. The fluoroscope may be of diagnostic aid when it shows a limit in the normal up and down movement of the heart during forced inspiration and expiration.

Pick's syndrome occurs in those cases of pericardial adhesions in which other serous membranes, the pleura and peritoneum, are also involved with inflammatory exudate (Fig. 55). To this polyserositis the term "pericarditic pseudocirrhosis of the liver" has been applied. The syndrome as defined by Pick consists of (1) a previous history of pericarditis; (2) enlargement of the liver; (3) obstinately recurring ascites; (4) absence of jaundice; (5) absence of signs of cardiac abnormality.

TREATMENT OF PERICARDITIS.

It should be distinctly borne in mind that the majority of cases of pericarditis, whether acute or with effusion, are self resolved under rest. When absolute rest is strictly enjoined upon the patient suffering from pericarditis but little other treatment is required for the condition. Puncturing of the pericardium (*paracentesis pericardii*) for the withdrawal of fluid is necessary only in the exceptional case.

Inasmuch as rest is the principal therapeutic requirement, drugs find little employment in the treat-



FIG. 55.—POLYSEROSITIS—PICK'S DISEASE.
Adjacent serous membranes are involved in the inflammatory process
of the pericardium. (Jefferson Medical College Museum.)

ment of pericarditis; they may of course be indicated in the treatment of the primary infection to which the pericardial inflammation is secondary. For example, if acute rheumatic fever be the causative condition, the use of the salicylates in divided doses which may range between 60 and 120 grains a day, would be indicated. Should tuberculosis be the cause of the pericardial effusion the treatment for tuberculosis is indicated. For precordial discomfort incident to pericarditis, an ice bag may be applied; it is not to be left continuously in place, but removed from the chest wall at frequent intervals. Pain, which is believed to be present only when the myocardium is involved, may require the sulphate of morphine hypodermically in quarter grain doses; the drug may also be necessary if the patient be anxious or restless. In the pericarditis arising during acute rheumatic fever, attention has been drawn by Billings to the almost specific action of cacodylate of sodium. Under its employment the exudate often rapidly disappears. The drug is administered hypodermically in doses of 1 to 5 grains which may be given from one to four times a day. To children a grain of the drug is administered every six hours.

It has been recommended in some quarters that a limited amount of exercise be given a patient suffering from pericarditis, in the belief that the exercise, by increasing the heart action, may prevent the formation of adhesions. This theory is mentioned here only to condemn it. In pericarditis the pericardium is relaxed as a result of inflammation and is not at the time capable of acting in its normal capacity—*i.e.*, as a supporting membrane for the heart. It is

obvious, then, that to impose upon the inflamed membrane the burden of increased heart action such as would result from exercise is only to pile Pelion upon Ossa.

Rest, absolute rest in bed, is the prime requisite in the treatment of pericarditis. It is to be continued not only during the activity of the condition, but also to be protracted beyond the period of convalescence. It is a matter of general observation that those patients who have had a long convalescence from diseases which induced pericarditis, are the patients who subsequently present the fewest number of symptoms suggestive of pericarditis with adhesions. When adhesions once form there is of course no drug or treatment which can in any way affect their presence. The time for treating adhesions is past long before they form. Should they form despite *absolute rest long continued*, the burden of their occurrence can then be laid upon the extent of the inflammatory process and not upon the shoulders of the attending physician.

Paracentesis of the pericardium, a procedure never to be lightly undertaken, may suggest itself in the presence of an effusion which is sufficient to embarrass the heart's action or to interfere markedly with respiration. Should such an effusion fail to improve from visit to visit and, on the contrary, increase, paracentesis should be performed. Large *purulent* effusions are of course to be evacuated; smaller purulent effusions that do not produce marked constitutional or mechanical pressure symptoms may present a fine question in treatment, as to whether they are better evacuated or better left alone in the hope of gradual and eventual reabsorption. The statistics of

operative interference in purulent pericarditis are not especially encouraging; Dolorme and Mignon have reported that in 80 instances in which paracentesis was performed, death resulted in 65 per cent. of the cases. Other statistics report a mortality of 40 per cent. as a result of the operation in purulent cases. It may not seem altogether fair to attribute such a mortality rate to operative interference, yet the figures indicate the seriousness of the undertaking.

When paracentesis of the pericardium is performed, the trocar may be introduced at any one of several areas, the site of election being in the 5th interspace, to the left of the sternum; the cardio-hepatic angle may, however, be the site selected. The skin of the patient is prepared as for any surgical operation, cocainized, and slightly incised: through the incision the trocar, guarded as to depth by the thumb and forefinger, is inserted with a deliberate thrust. If the patient can be propped upright in bed the position will facilitate the removal of fluid, which should be allowed to escape slowly. Should signs of collapse intervene the operative procedure will be stopped at once and treatment for shock instituted. The possibility that the fluid may be loculated is to be borne in mind: for this reason it may be necessary to enter the pericardial sac from more than one point in order to drain various areas to which laminæ of adhesions have confined the fluid. The wound is to be aseptically closed.

CHAPTER XV.

Myocardial Affections.

TERMS EMPLOYED.

THE musculature of the heart may exhibit either acute inflammation or chronic structural tissue change. To acute inflammatory processes in heart muscle which are quite constantly secondary to infections elsewhere in the body, the term *acute myocarditis* is applied. After the acute process has subsided the heart may be found to be permanently damaged as a result of the strain and infection to which it was then subjected. To such a circumstance the term *chronic myocarditis* is applied. Strictly speaking, the term is a poorly chosen one, for the heart muscle is not chronically inflamed; it is structurally altered. The damage usually takes the form of degenerative change or of fibrous tissue increase. The term chronic myocarditis also covers changes in heart tissue which result from long continued chemical poisoning or from degenerative change which results from long standing infections; but here again there is structural alteration, not inflammation of heart muscle in the strict acceptance of the term.

ETIOLOGY.

Acute Myocarditis.—Acute inflammation of the heart muscle is secondary to bacterial invasions elsewhere within the body. The *Diplococcus rheumaticus*
(189)

of Poynton and Payne, which is believed to be the cause of acute rheumatic fever is considered one of the most active agents in the production of acute myocarditis. Staphylococci and streptococci, particularly the *Streptococcus viridans*, are prominent etiological factors. The frequency with which the *Klebs-Loeffler bacillus* induces heart muscle change during the progress of or subsequent to diphtheria is a matter of general observation.* The sino-auricular block and higher grades of heart-block which burst from a clear sky and cloud the convalescence of diphtheria are evidence that the conduction system has shared in a more widely spread involvement of heart muscle. *Influenza* is an increasingly common cause of acute or sub-acute myocarditis and a particularly serious one, inasmuch as the damage to the heart muscle in sub-acute cases does not usually reveal itself until a considerable time has elapsed following the acute infection. Typhoid fever, scarlet fever and pneumonia are frequently attended with acute myocarditis, or it may arise during convalescence from these affections.

Measles, despite the fact that it has been lightly regarded as a "harmless" disease of childhood, intrudes itself with a persistence unusual to children's diseases, in the previous history of patients with myocardial damage. In writing of this subject from one of the early military camps in 1917 I presented the following:

"Measles, in these figures, assumes a percentage

* So far, my incompleated studies at the Philadelphia Municipal Hospital indicate that diphtheria may be expected to involve the heart muscle in varying degrees of severity, in 22 per cent. of all diphtheria patients.

importance far in excess of all the other acute, infectious, contagious diseases of childhood combined.

PREVIOUS HISTORY—DISEASES OF CHILDHOOD

	Per cent.
Measles	52.60
Diphtheria	9.79
Scarlet fever	15.00
Whooping cough	6.73
<hr/>	
Total of other diseases of childhood	31.52

"It would be reading entirely too much into these figures to assume from them that measles plays an important etiologic part in the cardiac changes of later life; it is, however, quite permissible to call attention to the earlier incidence of measles in over 52 per cent. of the patients with cardiac affections. That this traditionally "harmless" disease of childhood may be the precursor of eventual heart damage suggests itself in a plausible light when one reflects that the complications and sequelae of measles, such as middle ear involvements, pulmonic invasions and chronic catarrhal conditions of the upper respiratory tract, are frequently suppurative in character and hence as fully capable of affecting the heart as are other septic processes within the economy."

In this connection the question naturally arises "What is the usual percentage incidence of the diseases of childhood?" Their usual incidence can be approximated, but not accurately determined. There are insurmountable difficulties which rob any contagious disease statistics of their accuracy, such as a failure to recognize, a failure to report, or a desire to conceal; again, changes in population will alter the accuracy of figures. Admitting these and other

sources of error, an approximate incidence might be stated as follows:*

USUAL INCIDENCE OF THE DISEASES OF CHILDHOOD (Approximate).	
	Per cent.
Measles	18.1
Diphtheria	8.3
Scarlet fever	7.3
Whooping cough	3.3

In other words, it is probable that 18 out of a hundred people now alive have had measles, while the figures of those who had measles and who were later found to have heart affections are higher than this usual incidence by nearly three to one.

Further observations made in other camps confirmed the above figures. At Camp Custer, Michigan, measles appeared in the previous history of 88 per cent. of a group rejected on account of chronic myocardial change. In presenting these figures, it is not my intention at the present time to make any deduction or comment other than that *they are suggestive*.

"*Rheumatism*," in the present-day statistics of myocarditis does not loom so large as it did in the statistics of years gone by. The reason for this is, probably, that a distinction is now more generally drawn between acute rheumatic fever, which is a dis-

* *Basis for above percentages.*

These calculations are based upon statistics that show the number of reported cases in Pennsylvania for a 3-year period to be as follows:

Measles	73,486
Diphtheria	33,889
Scarlet fever	29,782
Whooping cough	13,341

During these years, the population of the State averaged seven million. The average length of human life has been figured herein as being 52 years.

tinct clinical entity, and the vague indefinite muscular pains and joint involvements which were hitherto included under the hybrid term "rheumatism"—conditions which are now believed to be the result of absorption from foci of suppuration—focal infection.

Chronic Myocardial Affections.—As previously stated, chronic myocardial change may ensue as a result of acute infections which involve the heart muscle. There are conditions other than acute infections, however, which induce structural alteration in the myocardium. Chemical poisons, such as lead, arsenic and mercury may, when absorbed over a period of time, produce myocardial change. Obesity has long been believed to be productive of chronic heart muscle alteration. Syphilis provokes changes of such a constant nature in heart muscle when seen at autopsy that the terms "syphilitic myocarditis" and "syphilitic heart disease" have come into existence as though to describe a clinical entity. Recent statistics indicate that syphilis was responsible for chronic myocardial change in only 9.7 per cent. of a group of cases studied. This, however, does not cover all of the instances in which syphilis might be an etiologic factor; for example, it is generally admitted that the *Treponema pallidum* is responsible for much of the damage that is done in diseases of the aorta. With this fact in mind, it is not difficult to understand how specific inflammation of the aorta might inflame the coronary arteries at their point of origin in the great vessel (see Fig. 75) and thus reduce the blood supply to the heart muscle itself, thereby bringing on chronic myocardial change; or by the extension of the aortic inflammation, through continuity of struc-

ture, to the coronary arteries these vessels themselves could readily become reduced in lumen and insufficient for the proper nourishing of the heart. An embolism or thrombus of the coronary vessels will also cause damage to heart muscle; should such an accident occur during the course of an acute infection it may produce sudden and unexpected death, or by a partial blocking of the blood supply, give rise to areas of degeneration in the heart walls.

Exophthalmic goiter, while an infrequent cause of myocardial change, at times affects the heart muscle, probably by the long-continued rapid action and stimulation of the myocardium which the elaboration of excessive glandular secretion may induce.

THE INDUCTION OF CHRONIC MYOCARDIAL CHANGE.

In tracing permanent heart muscle affections from their inception in acute myocarditis to their eventual development in chronic myocardial change, it is well to remember first of all that there are cases of acute myocarditis which to all appearances recover, either as a result of (1) cardiac resistance, (2) infections of a limited degree, or (3) through early recognition and skillful treatment. Aside from these instances, acute myocarditis induces permanent structural tissue change.

Rest, so necessary to a restoration of normal physical function, is a therapeutic measure obtainable only to a limited degree for affected hearts; and so, as the heart labors on, cloudy swelling and granular degeneration may affect its musculature; implication of the coronary arteries may starve the cardiac muscle

to an extent where ischemic atrophy ensues. Hyaline and fatty degeneration are unusual sequels to acute myocarditis, and yet they are found in post-mortem studies. In tissue under the microscope it has been



FIG. 56.—CICATRICAL MYOCARDITIS.

The photomicrograph illustrates the formation of scar tissue in heart muscle. *Practically the entire field is scar tissue; here and there appear muscle bands, marked "M." The dark spots are blood-spaces "B."* (Courtesy of Dr. Allen J. Smith.)

found that one muscle cell can be dissociated from its neighbor, each lying separate from the other. Fibrous increase of the connective tissue, usually distributed

irregularly through the heart, is a very frequent finding at necropsy (Fig. 56).

Fatty infiltration of the sub-pericardial connective tissue is often observed at necropsy. The thick fat is deposited not alone on the outside of the heart but also in the connective tissue between the muscle fibers. *Fatty infiltration* is to be distinguished from *fatty degeneration*, in which latter condition the fat is found in the muscle cell itself, which is a comparatively rare condition. *Fatty infiltration* is often suspected during life in cardiac patients who exhibit a general tendency to obesity. If there be an excessive deposit of fat in their bodily tissues it is perhaps a safe hazard to diagnose "fatty heart;" but there is no definite symptomatology nor are there any definite clinical signs by which such a condition can be recognized during life. Dyspnea and palpitation are symptoms of cardiac embarrassment in the obese but they are also symptoms of cardiac embarrassment in patients of slender build. Increase in the transverse diameter of the heart exists when it is subject to fatty infiltration, and it also exists when there is no evidence of fat deposit in heart tissue. Faint and muffled heart sounds occur in obese patients who might be suspected of "fatty" heart; and they occur with equal frequency in patients emaciated and exhausted by disease. There is no constructive evidence during the life of the patient upon which to base the once popular diagnosis of "fatty heart."

The pathology of myocarditis as outlined above, shows that such a possible variety of myocardial changes may give a variety of symptoms. It explains why acute myocarditis has no definite symptom-com-

plex. It explains why the diagnosis is more a matter of deduction than it is a question of physical signs. Many of the conditions just enumerated, such as cloudy swelling, ischemic atrophy, fatty infiltration, etc., cannot be recognized clinically, nor are they immediately incompatible with a fair degree of health. When they eventuate in chronic myocardial change, muscular weakness may not show itself until years after, when the reserve strength of the heart has been expended and cardiac bankruptcy is impending: then symptoms of heart failure are precipitated by some trivial incident or illness.

Cardiosclerosis is a term applied to sclerotic changes which take place in the musculature of the heart. There is no symptomatology or physical sign by which cardiosclerosis can be recognized during life; its presence can often be surmised when cardiac symptoms predominate in an arteriosclerotic process that is generally distributed. Cardiosclerosis is but another evidence of chronic myocardial change. It is produced by the same etiologic factors, it induces the same general symptoms of circulatory disturbance, and the symptoms may be benefited by the same methods of treatment, as any other chronic change in the heart musculature.

DIAGNOSIS OF ACUTE MYOCARDITIS.

Acute myocarditis is often difficult of recognition. It may be suspected when, in the course of a febrile condition due to bacterial invasion, there is detected an irregularity of the pulse which heretofore had shown no unusual change, especially if there be added to this a sense of exhaustion, out of all pro-

portion to the severity of the infection, *which is otherwise unexplained*. Hurried breathing, rapid pulse, cyanosis, pallor, coldness, fatigue or prostration induced by such trivial exertion as that of sitting up in bed, are further reasons for myocardial suspicion.

The detection of fine râles at the base of a lung posteriorly, particularly at the base of the left lung, which râles are not associated with other evidence of respiratory involvement, is often an early and always a valuable sign of myocardial affections. Such significant râles are not constantly present—they are evanescent in character, appearing with more frequency when the patient has been lying on the back for a few hours. Usually there are associated percussion phenomena, which consist of a deeper pitch to the percussion note, diminished resonance and an increased sense of resistance in an area which approximates that of the sixth, seventh or eighth dorsal vertebra, to the left of the spinal column. I am of the opinion that such percussion phenomena are occasioned by a left auricle that may be either relaxed in tone or increased in size. This chamber, it will be remembered, chiefly forms the posterior surface of the heart, and the pressure which it could exert, when for any cause expanded, upon lung tissue that offered little resistance on account of shallow breathing, would account for the production of râles at the base of the left lung in cardiac conditions.

Precordial oppression or precordial pain is of much significance. These symptoms may arise during the course of an acute illness, or they may arise late in convalescence; whenever precordial pain occurs a thorough search should be instituted for its cause,

whether the patient be bedfast or ambulant. Pre-cordial distress that may deepen into actual pain, is a symptom of heart affection that has not been accorded the degree of significance which its importance warrants; it may be of arterial or it may be of cardiac origin. I found it to have an average incidence of 77 per cent. in young men whom it was necessary to reject from military service on account of symptoms of chronic myocardial change. The physician should not lose sight of the fact that heart pain is also frequently referred to the neck or between the shoulder-blades and may be complained of more in these situations than at the precordium. An anatomic basis for heart pain as referred to the base of the neck may exist in the fact that it is from the first and second dorsal vertebrae that the heart receives its innervation during its embryotic development. Clinically, at least, the heart can so modify the irritability of the spinal segments of this area, perhaps reflexly, that heart pain is often referred to the lower cervical and upper thoracic nerves.

Palpitation and tachycardia may first direct attention to the heart in an acute infection with beginning myocardial involvement. Weak and irregular heart sounds may arise during the course of continued fevers, such as those which ensue during typhoid fever. In typhoid infection a gradual change takes place in the quality of the apical first sound which is best described by the term "snappy." It is due to the tautening of the mitral curtains, their action being more in evidence because, on account of muscular weakness, the "booming" quality of the first sound is faint or absent. If there be occasional reduplica-

tion of the first sound it is due to the fact that the impulse for contraction is delayed for a fraction of a second in reaching one ventricle or the other, probably owing to some inflammatory change involving the bundle of His. Systolic murmurs may be present at the base of the heart or they may be heard at the apex, in which latter situation they can be produced either by the stretching or the relaxing of the mitral ring (see Fig. 5, "Annulus fibrosus" or fibrous rings). It is quite likely, however, that murmurs which arise in acute myocarditis are for the most part produced by inflammatory changes in the endocardium, reflections of which help form the valves of the heart; for it is inconceivable that an inflammatory process could invade the musculature of the heart and not to some degree extend to the delicate and intimately adherent endocardium which lines the inner muscular wall.

The physician who fails to diagnose acute myocarditis should not censure himself too severely for a lack of diagnostic acumen. The indefinite and variable clinical picture, so often obscured or overshadowed by symptoms which are due to the primary infection, may readily cause one to overlook acute myocardial involvement.

Electrocardiographic Recognition.—Myocardial involvement may be suspected when an electrocardiographic curve reveals disturbances in the conduction system of the heart, as set forth in the concluding paragraph of Chapter XIII. Again, there are certain alterations in the waves of the electrocardiogram which indicate involvement of the fibers of Purkinje, (Fig. 43), and such disturbances have been subse-

quently found, at necropsies, to be definitely associated with hyaline, fibrous and other degenerative changes in heart muscle.

THE RECOGNITION OF CHRONIC MYOCARDIAL CHANGE.

Chronic myocardial change is usually the aftermath of acute myocarditis. The bacterial invasion which produced the acute condition is now no longer active; it has ceased to work acute inflammatory changes in heart tissue and has left as an aftermath degenerated areas of heart muscle, or has impaired the efficiency of the organ with an increase of interstitial connective tissue (Fig. 57). There may be just enough efficiency remaining in the damaged cardiac tissue to maintain the daily demands of a moderately active life. Sooner or later some extra burden is imposed upon heart structure that has no reserve force. It may be the extra burden of unaccustomed effort, or it may be a gradual reduction in the efficiency of one muscle fiber after another as the changes incident to increased labor or to the advance of years intervene, as in arterio- or cardio- sclerosis. It is then that chronic myocardial change becomes manifest.

Often, too, this condition is first suspected when there arises some enforced curtailment in the usual activities of the patient. Perhaps one in middle life becomes aware that he is no longer able to mount the staircase with his accustomed agility. He may notice that he is short of breath and that there is a sense of constriction about his chest as he hurries to his office. Faintness or dizziness may interrupt his game of golf or an unwonted feeling of exhaustion

and perhaps palpitation of the heart may follow the stimulation produced by motoring. With such mild limitations of accustomed heart response does early myocardial change announce itself.



FIG. 57.—FIBROUS MYOCARDITIS.

Areas of fibrous degeneration are excellently shown in the grayish patches of the heart muscle. (Jefferson Medical College Museum.)

Advanced myocardial damage is evidenced by the occurrence of the classical symptoms and signs of *heart failure*, in either moderate or pronounced degree. The symptoms of heart-muscle failure can be conveniently grouped under four heads, as follows:

(a) Early exhaustion attending trivial physical effort.

(b) Cyanosis and venous distension.

(c) Congestion of various viscera.

Visceral congestion produces a chain of symptoms and signs, depending upon the viscus involved, which are numerically set forth in the following paragraph.

Pulmonary congestion produces: changes in the respiration, varying from (1) hurried and shallow breathing to (2) dyspnea and (3) orthopnea; the presence of (4) moist râles in the lungs and other evidences of (5) chronic bronchitis; occasional (6) spitting of blood; sputum in which (7) "heart failure cells" may be found by the microscope. These are large, oval epithelial cells, presumably from the pulmonary alveoli; owing to long-continued passive congestion they contain blood-pigment granules of a brownish hue. They are also found in the sputum following pulmonary hemorrhage. *Renal* congestion produces: (8) edema; (9) ascites; (10) anasarca; (11) toxemia, deepening from mild uremic symptoms to (12) uremic coma; (13) ocular disturbances; (14) chemic and microscopic changes on urinalysis. There may also be present: (15) an enlarged and pulsating liver, or from this same cause (16) turgescient superficial veins; (17) painful engorgement of the spleen; (18) digestive disturbances.

The heart is usually increased in size. The pre-cordial impulse is often diffuse, and yet the ventricular impact is surprisingly lacking in the degree of force which one would expect to find. Basal systolic murmurs of varying degrees of intensity are frequently present.

(d) The *pulse irregularities* of heart failure may be nothing more than occasional and isolated *prema-*

ture contractions, although they are more often of the multiple and rapidly-recurring type. *Actual dropped beats* also occur and may be the precursors of deepening degrees of *heart-block*, with the *bradycardia* which usually (but by no means always) attends it. *Auricular fibrillation* frequently sets in, more often as a permanent than as a transitory condition. Brief periods of *tachycardia* are not at all uncommon. *Pulsus alternans* may be observed over a period of months, although its occurrence is generally premonitory of the approaching end of life in chronic myocardial degeneration. In short, any of the pulse irregularities mentioned in Chapter XIII may be present in myocarditis, for such irregularities are, for the greater part, simply evidences that the conduction-system of the heart has shared in the process which affected the heart muscle.

TREATMENT OF ACUTE MYOCARDITIS.

The treatment of acute myocarditis is *absolute rest in bed*. Acute myocarditis continues acute as long as the causative factors are active. The best hope of lessening the degree of cardiac damage which is induced by the causative bacterial invasion, toxin or chemical poison lies in relieving the heart of as much effort as is possible in order to increase its rest period; hence the dictum, *absolute physical rest*.

The value of rest cannot be over emphasized: if by absolute rest in bed the physician can secure a reduction of twelve beats per minute from a heart-rate of 120 in acute myocarditis, in 24 hours he has saved the laboring organ 17,280 cycles. In other words he

has given to the heart over 4 hours of additional rest out of the twenty-four by the prolongation of diastole, at the expense of which rapid rates are maintained.

In addition such measures may be employed as are detailed under the treatment of acute endocarditis in the following Chapter XVI. Treatment for the one acute condition is quite the treatment for the other, for one cannot suppose the existence of an acute myocarditis which has been induced by infections, without presupposing the co-existence of endocarditis to some degree, owing to the intimate association of these two structures.

TREATMENT OF CHRONIC MYOCARDIAL CHANGE.

The physician does not see chronic myocardial change at its inception; he sees it after it has been established for a period of time and become distinctly noticeable as the immediate consequence of an extra burden recently placed upon the heart. Logically the endeavor of treatment should be to first remove the *extra* load, and this includes an *unremitting search for a focus of infection* (page 224) that may be aggravating the heart's distress: next, to compensate for the extra burden by relieving the heart from even its accustomed daily effort for a period of time and finally, to guard against future strains on the myocardium by proper instruction of the patient (see *The Patient's Daily Life*, page 370). The first two indications are met by rest and other appropriate treatment which can be set forth as follows:

INDICATED TREATMENT.

1. Rest—physical.
2. Rest—mental.
3. Rest—emotional.
4. Elimination.
5. Improvement of heart nutrition.
6. Alleviation of incidental distress.
7. Sustaining of the heart with drugs.

(1) *Physical Rest*.—At the beginning of treatment it is a good rule to put the patient to bed. While this may seem too arbitrary a procedure to some patients, it is much easier for the physician to lessen restrictions as circumstances may permit rather than to later impose added restrictions upon a patient who has been allowed to be ambulant. As the symptoms which caused the patient to seek medical advice improve, the liberty of the room or house may be allowed. The period of confinement to bed is a variable one and can be determined only by the disappearance of cardiac symptoms with no unfavorable signs manifesting themselves on attempted effort; the patient may then be permitted to undertake simple physical effort. If the pulse shows a marked change in rate when the patient is first permitted to be out of bed it is an indication that the period of rest has not been sufficiently long continued. A change in rhythm may not be of the same significance as a marked change in rate, for an alteration in rhythm is frequently encountered as a previously-established condition in patients of advancing years who have chronic myocardial change. Again, such alteration in rhythm may have just had its inception during the recent collapse, threatened or actual, which caused the physician to put the patient to bed and be, from this

time on, permanently established. But if a change in rhythm or an alteration in volume is *brought on* by a change in posture or by the trifling effort of attempted locomotion, the patient should be remanded to bed. Judgment dictates, of course, that the aged and the infirm who bear bed confinement very poorly as a rule, should be permitted a limited amount of liberty, of which liberty they are very often the best judges, rather than to be fretted and annoyed by a too arbitrary change in their accustomed mode of life.

(2) *Mental rest* is of but little less importance than is physical rest. This is frequently demonstrated by the beneficial results which follow institutional treatment in sanitarium or in hospital. There, for example, the head of a household is free from the worry of conducting or superintending a home, rid of the vexation of servants, away from the intrusions of over solicitous friends, and is amid quiet rest-inducing surroundings where the ear is not strained to catch every unusual sound nor the mind kept busy interpreting them, as would be the case were the patient abed at home. To many patients, particularly those of an anxious turn of mind, there is much mental ease afforded by the thought that in a sanitarium a physician is always within easy call; and from the point of view of the family doctor there is much advantage in the medical and nursing supervision, in the scientific preparation of meals, in the regulation of rest and in the other many attentions which spell efficiency in modern institutional management.

However, treatment at an institution is not advisable for every case of cardiac disease. Patients with marked edema, cyanosis and dyspnea are often

better treated at home, particularly if the institution be a long distance away and imposes the effort and fatigue of a protracted journey upon the heart which is already showing symptoms of exhaustion.

(3) *Emotional disturbances* are difficult to eliminate in the home management of a cardiac patient, but they should be guarded against in every possible way, for at times they have a profound influence upon the affected heart. The attentions of home nurses may irritate; the sympathetic eye of a friend who has been permitted to see the patient may cause depression of spirits; a whispered voice may be interpreted as ominous by the invalid whose sole thought is on her physical condition, or a laugh may be mistaken for an utter lack of sympathy. In such ways may the emotions of a patient be played upon and, through the sympathetic nerves, actually play upon the heart.

(4) *Elimination* will need to be practised with more or less constancy in the treatment of the patient who is confined to bed. When *the bowels* are to be stimulated over a period of time it is better to use the milder laxatives in daily dose, rather than to employ drastic cathartics at intervals; for the latter, by depleting the system through excessive action, may add the burden of physical exhaustion to a laboring heart. The mild laxatives such as cascara sagrada, senna, compound licorice powder or the saturated solution of magnesium sulphate in small daily dose are therefore the evacuants of choice.

The skin can be kept sufficiently active by the daily tepid bath followed by witch hazel rubs, and aided by the employment of massage. The induction of free perspiration is to be avoided for it may prove very

exhausting to the patient with heart muscle damage. Of course the urgent incidence of uremia may demand hot packs; or the occurrence of apoplexy may necessitate prompt and vigorous catharsis; but these are exceptional instances in the treatment of moderately advanced myocardial affections and are, indeed, two of the many possible occurrences which the physician hopes to avoid when he puts the failing patient to bed.

The kidneys can be kept in a freely eliminating state by the drinking of water in liberal quantities. As concerns the use of various mineral waters which are supposed to be active renal eliminants; they seem to possess little virtue other than that which could be ascribed to mechanical flushing. However, if the patient, urged by a friend's advice feels that these waters have some curative virtue, the physician has nothing to lose in permitting their employment; patients frequently drink such waters with more regularity and faithfulness than they accord to ordinary unheralded and unlabelled water, and the physician's purpose of renal flushing is thus secured.

Infusion of digitalis may be used where a more active renal stimulant than plain water is desired. The infusion of digitalis should be prepared by the pharmacist; in order to extract the active principles of fox-glove leaves it is necessary that a small amount of alcohol be used in preparing the infusion. Certain glucosides upon which the activity of digitalis principally depends, *viz*: digitoxin and digitalin, are freely soluble only in the presence of alcohol. The usual aqueous preparation made by pouring boiling water on the leaves is inert so far as definite digitalis

effect is concerned. The only really active one of the five digitalis glucosides which such an aqueous solution contains is digitalain.

(5) *Improvement of heart nutrition* may be accomplished in an indirect manner by easing the load and by increasing the period of cardiac rest through lessened physical effort. As to the *direct* effect of foods upon heart muscle, much yet remains to be discovered. A cardiac patient who is at rest in bed or confined to his room manifestly requires less nourishment than when he is up and around; consequently he must reduce the intake of food if he would avoid overloading kidneys, bowels and liver and by remote effect on these organs indirectly aggravate the condition of the heart and circulation. Small quantities of nutritious foods prepared in their most easily assimilable form, together with the limiting of protein intake, and with the elimination of foods which are known to be productive of intestinal fermentation in a given case, are more desirable rules to follow than is the blind adoption of one of the many "cardiac diets" which appear from time to time. A standard diet is given for reference in Chapter XXII.

One notable exception to the general impracticability of rigid food regulation in cardiac patients is the diet suggested by Karell, to be used when it is desired to limit the *water intake*. There are instances where unlimited water intake might add to an existing edema, ascites or other effusion and thus add to the embarrassment of the heart. In Karell's diet fluid intake is limited to milk; the free use of salt is interdicted, on the theory that it accumulates in the tissues and attracts fluids to the parts. Even sufficient

salt to maintain the normal individual demand of 15 grains per day is prohibited at first, until the excess previously stored in the tissues may be considered exhausted. The regime is as follows:

The Karell Diet.—For the first seven days, 8 ounces of milk at 12 A.M., 4 and 8 P.M. No other fluid.

Eighth day, milk as above, and at 10 A.M. one soft boiled egg; at 6 P.M., two pieces of dry toast.

Ninth day, milk as above, and at 10 A.M. and 6 P.M. one soft boiled egg and two pieces of dry toast.

Tenth, eleventh and twelfth days, milk as above and at 12 noon chopped meat, rice boiled in milk, and vegetables; at 6 P.M., one soft boiled egg.

No salt is used throughout the course. Toast and butter are to be salt-free. A small amount of cracked ice is allowable. All meat is advantageously omitted.

When the desired effect has been attained by this dietetic regime, the effect is maintained by an occasional "Karell day," in which the strict food regulation of the first seven days is adhered to for 24 hours.

Cane-sugar in Heart Affections.—The classic experiment of F. S. Locke,¹ in which he demonstrated that the excised heart of a rabbit could be kept pulsating for four days by pouring through it a solution containing dextrose, has stimulated much interest in the clinical value of sugar as a means of nourishing depraved heart-muscle. Physiologists have announced that the sinoauricular node, the bundle of His, and its arborizations, contain a remarkable amount of glycogen. Prof. Dr. Adamkiewicz² states that the heart requires its own weight of sugar each day for its

¹ Locke: Zentralbl. f. Physiol., No. 20.

² Adamkiewicz: Prag. med. Wchnschr., No. 43, p. 601.

nourishment (9 to 11 ounces). These observations have been utilized by Sir Arthur Goulston, of Exeter, in the treatment of heart affections with cane-sugar, carefully avoiding the employment of beet and any sugars other than cane. He begins with the administration of 2 ounces a day, pushing the administration rapidly to 4 ounces a day, or even to 10 ounces in some instances. The likelihood of digestive disturbances and intestinal fermentation is disposed of by the observation of Abderhalden¹ that the lactic acid ferment of the intestines does not attack cane-sugar or milk sugar. Sir Arthur reports brilliant results following this plan of treatment.

Breathing exercises seem to have a beneficial effect in improving the nutrition of the heart in some patients. Their employment is based upon the theory that slow and deep expiration will so decrease intrathoracic pressure upon the heart that the heart muscle itself will receive a greater blood supply under such a circumstance than during ordinary quiet breathing. By the same reasoning, slow and deep inspiration will decrease the intrathoracic area occupied by the heart and thus by compression more completely empty the heart muscle of venous blood and permit the ingress of oxygenated blood with the ensuing inspiration. Whether the theory appears chimerical or not, the fact remains that some patients have an increased sense of well-being and seem benefited as a result of the practice, which certainly has to commend it a more liberal aëration of the blood than is secured in ordinary quiet breathing. There are foreign physicians who employ a device to stimulate deep breathing in their

¹ Abderhalden: Physiological Chemistry, Lecture XX.

patients who suffer from chronic myocardial change, the device consisting of two bottles connected to one another by a piece of glass tubing, one of the bottles being perhaps half filled with water; a length of rubber tubing is attachable to a glass tube which is inserted through the cork of both bottles. The exercise consist in the patient blowing through the rubber tubing and attempting to force the water from one bottle into the other, with as few breaths as possible. Add water as capacity of the patient's lungs increases; interest in the procedure is thereby maintained.

(6) *Alleviation of Incidental Distress.*—Distressing circumstances which add to the heart load and which the physician may have to ameliorate, are: anasarca, pain, sleeplessness, constipation, hepatic torpor, dyspnea, bronchitis, and vomiting. Where possible, remedial measures other than drugs should be employed. Dropsical effusions may be relieved by the trocar and cannula; painful engorgement of the extremities by Southey's tubes or multiple punctures ($\frac{3}{8}$ inch deep) of the tense and edematous skin, always under antiseptic precautions and with subsequent aseptic dressing; pain may be amenable to the ice-bag or hot fomentations; sleep may be induced by hot drinks, by massage or friction rubs; constipation and hepatic torpor frequently yield to a diet of laxative foods or enemata; dyspnea, to a change in position, or the use of oxygen; bronchitis, to the gradual improvement of the cardiac condition; and vomiting, to a temporary withdrawal of all foods by the mouth, save the sipping of ice-water, and to the counterirritant effect of mustard plasters (1 part mustard, 5 parts flour) applied to the epigastrium.

(7) *Sustaining the heart with Drugs.*—There is no specific drug treatment for chronic myocardial change, drugs being employed only for the treatment of symptoms as they arise and only when the simpler measures just described are not effective. Digitalis is a remedy very often abused by being promiscuously administered. In many quarters it seems to be the first thought when a cardiac or circulatory disturbance is detected. Digitalis is by no means a panacea for all cardiac ills. There are some cardiac conditions which are made distinctly worse by its indiscriminate employment. By calling forth *all* of the scanty stock of residual effort that yet remains in a seriously-damaged heart, digitalis can bring on death by utter myocardial exhaustion. In chronic myocardial affections there are two indications which warrant the use of tincture of digitalis: either *a failure of the heart muscle to improve in tone* under rest and other effort conserving methods, or else *a progressive weakening of the heart muscle* despite rest and other effort conserving methods. Under these circumstances the “average dose” of 8 minims of the tincture, repeated 3 times a day, is quite often sufficient. The drug should be withdrawn when the desired effect is produced. (See Untoward Effects of Digitalis, Chapter XXIV).

The x-ray may have a therapeutic value in myocarditis in those rare instances where the disease can be attributed to no other cause than excessive glandular activity, as exemplified in thyroid disturbances. A Röntgen light over the thyroid may reduce activity of the gland and, by lessening the excessive elaboration of secretions, lessen their effect upon the heart.

CHAPTER XVI.

Endocarditis.

THE DEFINING OF ENDOCARDITIS.

THE term endocarditis in its customary acceptance implies acute or chronic disease of a heart valve. If one does not amplify this limited use of the term one may not secure a clear picture of the damage which is wrought in the lining membrane of the heart during the progress of or subsequent to acute infections elsewhere within the body. It should be remembered that while acute inflammation of the lining membrane, from the standpoint of gross pathology, is *usually* located on the valves, yet such is not always the case. It may be mural and distributed quite generally over the interventricular septum and even involve the chordæ tendinæ and the muscoli papillares.

It is questionable whether acute endocarditis ever *confines* itself to the valves and the mural endocardium alone. One of the symptoms of the condition and one which often first draws attention to the heart, is irregularity of the pulse. Irregularities of the pulse, for the greater part, reflect disturbances of the conduction system. The conduction system of the heart is distributed in the muscular tissue of the heart walls; hence it is reasonable to assume that any inflammation of the *endocardium* which produces an irregular pulse must have penetrated that thin translucent membrane and have reached to the heart muscle itself.

Acute endocarditis is quite invariably a secondary process. The degree and extent of the cardiac inflammation depends upon several factors. For example, certain bacteria are more virulent than others and can be expected to set up a greater inflammation in the lining membrane of the heart; as the infection is probably from the blood stream, the *numbers* of bacteria which might be present would also have an effect upon the degree of inflammation; and, if the cardiac tissues have been *lowered in resistance* through an exhausting primary illness or have been weakened by previous attacks of disease, an added endocardial inflammation will be the more serious in consequence.

The varieties of endocarditis are for the most part arbitrary distinctions, which are justified in that they enable easier descriptions of the various processes. But it should be remembered that when inflammation of the lining membrane of the heart sets in during an acute infection there is no way of telling whether it will be "simple" or malignant, whether its course will be acute or chronic. The acute may suddenly become malignant; and the malignant, which at first threatens to overwhelm, may as suddenly modify its form and become the protracted variety of malignant endocarditis,—that type of slow evolution, the principal cause of which is now believed to be the *Streptococcus viridans*.

We have then, for convenience of description: (1) acute "simple" endocarditis; (2) acute malignant endocarditis and (3) chronic malignant endocarditis. In any of these three forms the causative agent, bacterial infection (perhaps rarely a chemical poison), is

actively present; not until that activity has ceased and the cause become quiescent, does (4) *chronic* endocarditis ensue. Strictly speaking, "chronic" endocarditis is a poorly chosen term, for there is no actual inflammation present in the endocardium; a previous inflammation has terminated in structural tissue change and the resultant permanent damage in valve structure is better known as *chronic valvular disease of the heart*, a subject which is discussed in the chapter which follows.

Endocarditis, on account of its frequent association with acute rheumatic fever, is sometimes spoken of under the misleading term of "rheumatism of the heart." The term should be discarded; it gives no clear conception of the condition and may utterly misdirect the treatment of the physician who relies upon "antirheumatic" drugs alone to correct the perverted cardiac condition.

MORBID ANATOMY.

Acute Endocarditis.—Early cases of endocarditis which come to post-mortem exhibit on the valves small, light colored beads which are arranged in a crescentic form, a line or two from the free edge of the valve, at that point where the leaflets touch each other when closed. These small beads are composed principally of fibrin in which micro-organisms can be found. To the unaided eye the walls are not as yet involved. Later on, as the inflammation progresses, these beads assume the shape of cauliflower-like excrescences and to this form the term "verruccose endocarditis" is applied by the pathologist (Fig. 58). Eventually the process involves the deeper tissue of

the valve, then spreads to the valve-bases and from there to the heart wall itself (Fig. 59). It has been observed that when the aortic valve is seriously invaded the infection has a tendency to spread in the



FIG. 58.—VERRUCOSE VALVULITIS.

Close inspection of the leaflets will reveal the grayish, pin-point deposits which are the first macroscopic evidence of valvular disease. (University of Pennsylvania Medical School Museum, collection of *Dr. R. S. Willson*.)

direction of the aorta; profound infection of the mitral valve spreads upward to the auricle and down in the ventricle to the chordæ tendineæ, and to the muscoli papillares.

Acute endocarditis is much more common on the left side of the heart than on the right. For this reason the mitral and aortic valves are found at necropsy to be the ones most frequently affected; it is unusual for all four of the valves to be involved. Endocarditis may occur during intrauterine life in



FIG. 59.—VEGETATIVE MURAL AND VALVULAR ENDOCARDITIS.

The black arrow points to the inflammatory process which involves the heart muscle wall as well as the valve above. The white arrow indicates further mural endocarditis. (University of Pennsylvania Medical School Museum.)

which event the valves of the *right* side are those which are the most often affected. From this cause some forms of congenital heart disease arise.

Malignant endocarditis presents a picture of roughened valve surfaces which become the seats of fibrin deposits whipped from the blood stream; these

deposits become infected and may be dislodged and swept as *emboli* to other parts of the body (Figs. 60, 61 and 62). Retraction of the leaflets may occur and necrosis, ulceration and perforation of the valves be a part of the necropsy findings (Figs. 63, 64 and 65). It has been observed that two or more valves are in-

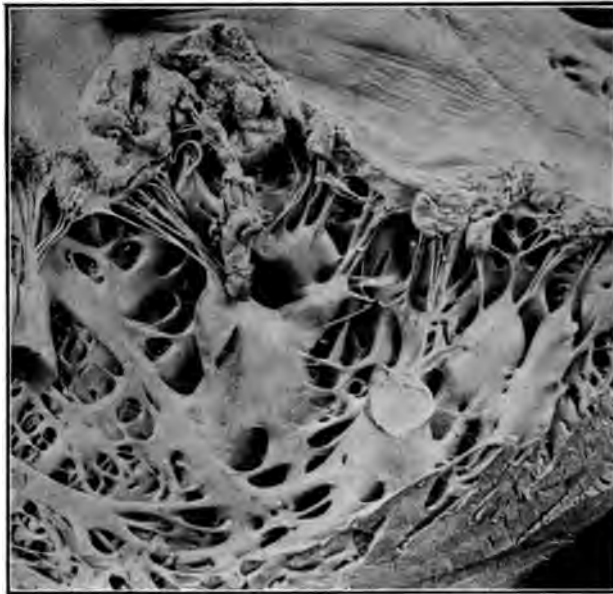


FIG. 60.—MITRAL VALVE VEGETATIONS.
(University of Pennsylvania Medical School Museum.)

involved in less than half of the cases of malignant endocarditis which come to necropsy and that the mural endocardium is invaded by the process in over one-fourth of the cases so studied. The myocardium and pericardium may also be affected, constituting a *pancarditis*. Especially is this true of the streptococcic heart affections of children.

ETIOLOGY OF ACUTE ENDOCARDITIS.

The affection occurs for the most part in childhood and in adolescence, although it is not confined to early life. Its frequency in youth is explained by the frequency of acute rheumatic fever during that period of existence. In a series of 173 cases of acute



FIG. 61.—AORTIC VALVE VEGETATIONS.
(University of Pennsylvania Medical School Museum.)

rheumatic fever it was found that 53 per cent. of the patients had acute endocarditis. In this connection it is well to recall that the "growing" pains of children are very often a manifestation of rheumatic fever or of absorption from a septic focus. In a series of cases of acute rheumatic fever associated with endocarditis which came to the post-mortem table, it was found

that the mitral valve alone was affected in 85 per cent. of the cases; the aortic alone in 3 per cent.; and that both mitral and aortic valves were involved in the remaining 12 per cent. of autopsies.

Tonsillitis is so frequently followed by acute rheumatic fever or by arthritic involvement that it is per-

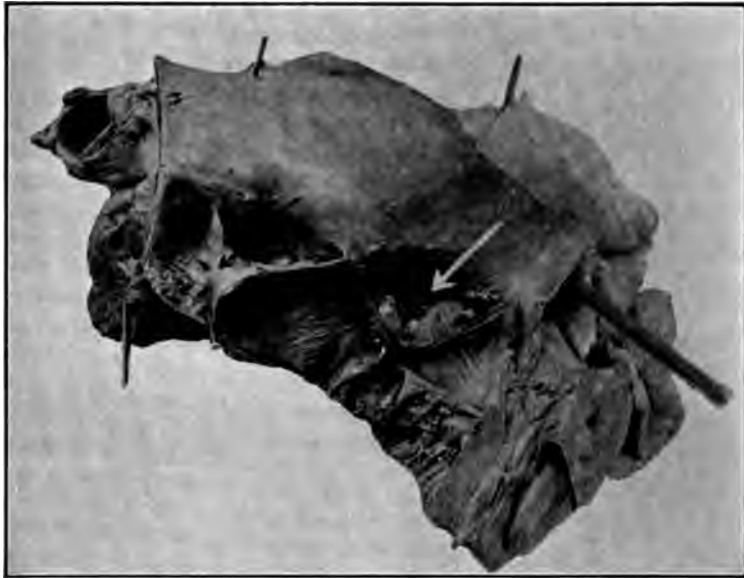


FIG. 62.—FIBROUS FUSION OF VALVES.

The black arrow points to the fibrous fusion of two aortic leaflets. The white arrow indicates sclerotic and calcified vegetations within an aortic cusp. (University of Pennsylvania Medical School Museum.)

haps the second most frequent cause of acute endocarditis.

Chorea is very frequently accompanied with or followed by endocardial infection. Of 171 cases of chorea which came to autopsy endocarditis was found in 90 per cent. of the total number. *Pneumonia* was

found by Osler to be accompanied by endocarditis in 16 per cent. of the cases of pulmonary disease which came to necropsy. *Typhoid fever* gives an incidence

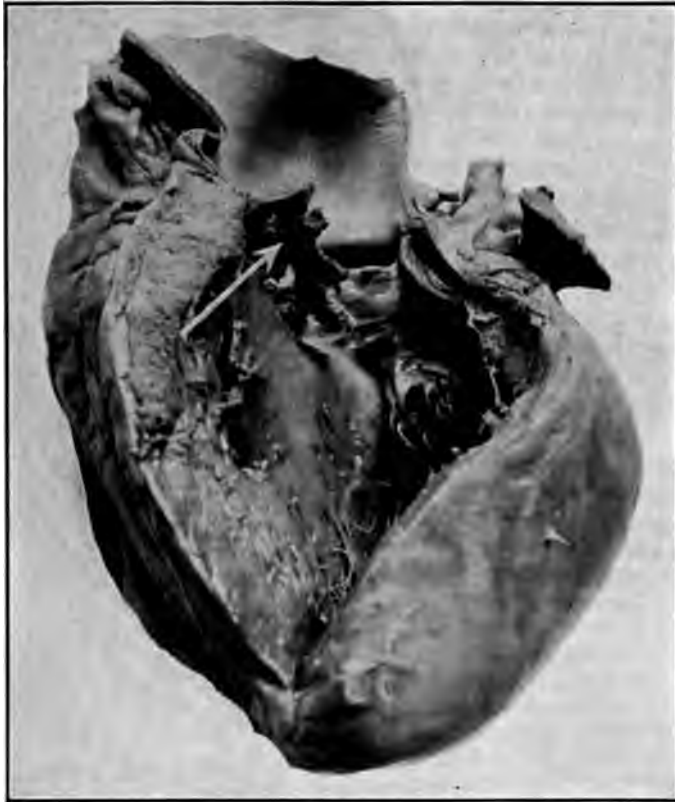


FIG. 63.—AORTIC VALVE LEAFLET DISTORTED BY VEGETATIONS.
(University of Pennsylvania Medical School Museum, collection of Dr. R. S. Willson.)

of 12 per cent. of endocardial infection; *scarlet fever*, according to my observations, less than 3 per cent. *Erysipelas*, *osteomyelitis*, *infected wounds* and *puerperal fever* have been found to be associated with endocarditis.

Focal Infections.—The percentage of endocardial inflammations which arise as a result of focal infections is a difficult one to calculate. It not infrequently happens that no other cause can be found for endocarditis than a focal infection which, although it may

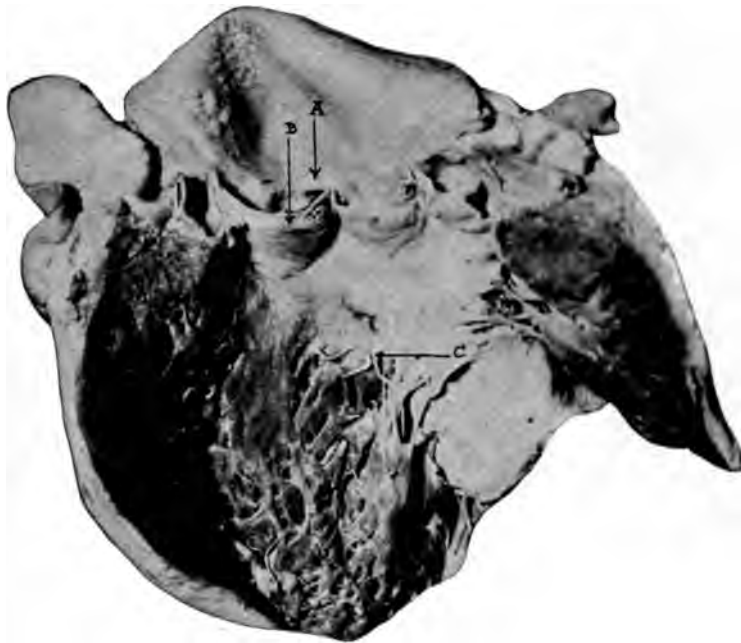


FIG. 64.—FENESTRATION OF AORTIC LEAFLETS.

The black arrow "A" indicates the fenestration. Despite this opening, the valve probably was not insufficient, for the fenestration occurs along the free edge of the valve, *beyond the line of closure*, as indicated by the arrow "B." Arrow "C" points to an aberrant chorda tendinea. (University of Pennsylvania Medical School Museum.)

have been comparatively quiescent for some length of time, becomes active when the powers of resistance of the individual are for some reason reduced; bacterial infection then gains ingress to the circulation and

affects the lining of the heart. For that reason the physician should institute a thorough search for foci of suppuration which may arise from dental sepsis, infective tonsils, occasionally in chronic forms of ear

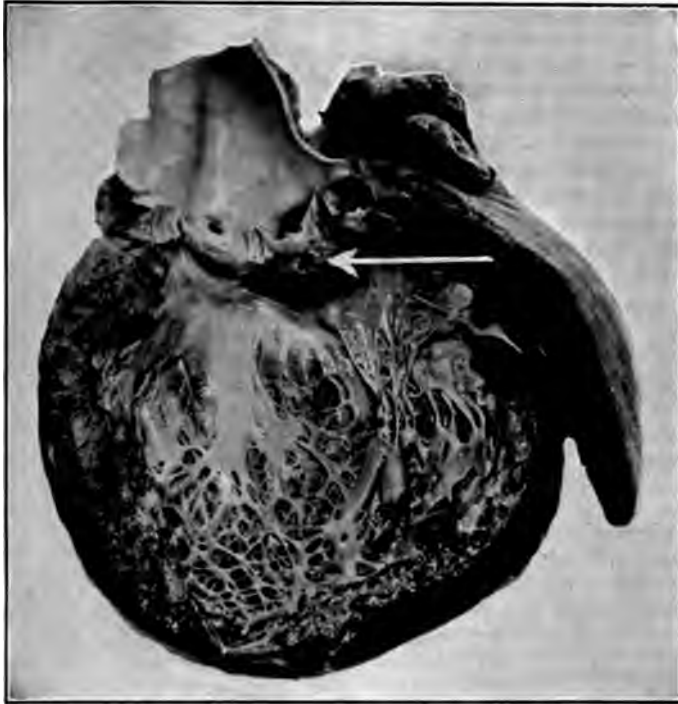


FIG. 65.—PERFORATION OF AN AORTIC LEAFLET.
(University of Pennsylvania Medical School Museum, collection of *Dr. R. S. Willson*.)

disease, and less often in gall-bladder infections, perirenal abscesses, pyelitis, prostatitis and chronic inflammations of bones or joints.

Foci of suppuration which are often overlooked may be found in apical abscesses of the teeth (Fig. 66). The physician should not be satisfied with the

statement of the patient that his teeth were recently pronounced to be in a healthy condition. A negative report from the dentist should not disarm the suspicion of a dental cause when the conviction has been once established; only upon the receipt of a negative *x*-ray examination, in which the *entire denture* has been photographed, should abscessed teeth be ruled from consideration. The teeth should not be excluded as possible foci of suppuration simply because there is an absence of pus-pocket shadows on the film. Bliss¹



FIG. 66.—APICAL ABSCESS.

The abscess at the apex of the tooth gave no sensations whatever of pain, elongation or tenderness. It finally ruptured through the skin below the chin, and the discharge of purulent material was believed by the patient to be due to a "wild hair," for he had been assured repeatedly that his teeth were in excellent condition.

informs us that at the first examination the Röntgenologist may find only some thickening or irregularity of the peridental membrane, but that an exposure made at a later date may show a slightly darker area around the apex of the tooth, indicating that absorption has taken place. *Capped teeth* are to be regarded with suspicion, whether or not they give sensations of elongation, pain or tenderness.

Of tonsillar conditions it may be said that the chronically inflamed tonsil is the one likely to cause

¹ Bliss, Gerald D.: Penna. Journal of Röntgenology, Jan., 1917, p. 9.

endocarditis. Whether this be a catarrhal inflammation or whether the crypts of the tonsil present caseous patches—follicular tonsillitis—makes little difference; whether the tonsils be hypertrophied or submerged makes little difference; if the tonsils are chronically inflamed or frankly diseased they should be removed. A *diseased* tonsil is always a *potential*, and sometimes an *actual* cause of endocarditis, and the mere fact that it is not a normal gland and that it is subject to occasional or to repeated attacks of acute inflammation is sufficient cause for its enucleation. Removal of part of the gland by clipping it—unsympathetically referred to as “massacre of the tonsil”—is worse than useless, in that it creates a false sense of security against future inflammatory processes. When tonsillectomy is indicated, the indication is for complete enucleation, not a vestige of the gland being permitted to remain.

ETIOLOGY OF MALIGNANT ENDOCARDITIS.

While it is quite possible for malignant endocarditis to be caused by the same factors which produce the acute form, yet as a matter of clinical observation, the malignant type has *an added etiology quite its own*. It arises for the most part in individuals who have had *previous* inflammation of the heart valves which permanently damaged the leaflets, anteceding by perhaps many years the occurrence of malignant endocarditis. For this reason malignant endocarditis is much more frequent in adults than it is in children, it having been shown that in patients suffering from the disease 65 per cent. are 25 years of age or older.

Septic mouth conditions are believed to be factors of no small moment in the production of *malignant* endocarditis. Dental caries, apical abscesses, pyorrhea or other foul conditions of the mouth very often harbor the *Streptococcus viridans*. This is the streptococcus which is most frequently found in subacute or chronic malignant endocarditis, and it has been cultured *post mortem* from vegetations which have been found on the heart valves. Other staphylococci and streptococci have also been found to be etiologic factors in malignant endocarditis. The essential point to remember is that the condition is usually the result of a bacteremia; the blood infection finds a point of less resistance on valves which have been previously damaged, either as a result of inflammation many years gone by, or as a result of recent acute infections; or still again, bacteria may find the endocardium lowered in resistance by long continued septic absorption and hence a vulnerable point for attack.

It is unfortunate for the bacteremic point of view that blood cultures are so often sterile in malignant endocarditis. Certainly they seem to be more uniformly negative in the early period of the disease, at the very time when they could be of the most value in diagnosis and in treatment, than they are later in the condition when the patient is desperately ill. There are some bacteriologists who have a peculiar aptness in finding bacteria in the blood and it is a point of repeated observation that one with this aptitude can find the bacterium when others have failed. One such who had the happy faculty of demonstrating the organism with remarkable ease, told the writer that the blood cultures were more likely to be positive if

the blood were taken just before the anticipated advent of a septic chill or immediately following symptoms suggestive of an embolism having lodged. The statement is given here for what it may be worth; if it aids in one single instance in the early recognition of the organism and thus facilitates the early preparation of an autogenous vaccine, it will have been well worth repeating.

SYMPTOMS OF ACUTE ENDOCARDITIS.

The symptoms of acute endocarditis may be overshadowed by the severity of the primary infection or they may be overshadowed by the pain of a primary arthritis. *Fever* is usually present, but it is of no particular type and is difficult to distinguish from the fever that is due to the primary condition. The physician should take pains to routinely examine the heart of a patient who is ill with an acute infection, for attention may thus be early drawn to changes in the heart sounds which take place from visit to visit. These changes may gradually develop into *murmurs*. Such murmurs are usually at the apex of the heart, although they may be basal; they are systolic in time; in quality they are at first soft, but eventually become loud and gross, although during the period of valve roughening they are often of musical quality. The musical note may be observed to recur from time to time, probably due to a vegetation which has formed on a valve and projected itself into the blood stream at an angle sufficient to produce a sound of musical timbre. The murmurs of *acute* endocarditis are at times evanescent in character and a murmur distinctly audible at one examination may not be heard

at all at the next. The *pulse* often has a rate out of all proportion to the fever and to the discomfort of the patient. This increased pulse rate may continue throughout convalescence from the initial illness. The occurrence of an *irregular pulse* announces some disturbance of the cardiac mechanism which for the most part takes the form of premature contractions or of auricular fibrillation. Dropped beats or higher grades of heart-block may occur, although such blocks are usually of a transitory nature. In this connection it might be well to state the self evident truths that *murmurs are quite invariably the result of ENDO-cardial conditions. Pulse irregularities are quite invariably the result of MYO-cardial conditions.*

PHYSICAL SIGNS OF ACUTE ENDOCARDITIS.

On *inspection* one may note a degree of respiratory embarrassment in acute endocarditis. It may further be observed that the apex gradually moves toward the left as the days pass. An over-acting heart often exhibits a precordial impulse which is widely distributed over the left chest. To an impulse of such extent the term *irradiation* is applied.

Palpation will confirm the latter observations. The pulse is often rapid and irregular and may show wide variations in rate due to change of posture of the patient. The mere act of turning over in bed may cause an acceleration of perhaps ten beats a minute and the patient seem exhausted as the result of the trivial physical effort.

Percussion is of little value in acute endocarditis. If one has made a careful record of the transverse diameter of the heart on the occasion of one's first

visit one may, by comparing this record with the observations of subsequent days, detect an increase in the transverse diameter of the heart. Percussion is also of service in drawing the attention of the examiner to a pericardial effusion which may complicate acute endocarditis; especially is this likely to happen in the streptococcic infections of children.

Auscultation.—By auscultation may be frequently noted a gradual change in the character of the heart sounds. The first sound at the apex alters in quality and intensity; it may be “blurred,” “muffled” or “prolonged,” and from these varying degrees of change eventuate into a murmur, usually located at the apex, always systolic in time. At first the murmur is soft and blowing, and the second sound of the heart as heard at the pulmonic area is accented; later on the murmur may take on the rough, loud or occasional musical quality previously mentioned.

It is neither the presence of a murmur nor its pitch nor its intonation that forms the suggestive diagnostic feature in acute endocarditis. It is the *sequence* in the *alteration* of *heart sounds*—the gradual evolution of normal sounds into a murmur—that is of diagnostic significance. It should be remembered that systolic murmurs can be variously produced (page 78) and that those found to be present at one visit are absent perhaps at the next. Such murmurs of course do not have the significance attached to them which can be attached to a murmur which gradually evolves and which is the direct result of valve invasion.

We have seen under morbid anatomy that the mitral valve is the valve which is affected in 50 per cent. of the cases of acute endocarditis which have

come to autopsy. While lesions of the mitral valve may produce either a systolic or a presystolic murmur, one should not lose sight of the fact that an apical *pre-systolic* murmur occurs only in mitral stenosis. Mitral stenosis is the result of long continued valve change, and therefore one would not at all expect to hear a *pre-systolic* murmur in acute endocarditis, where the murmur develops in a few days and where the valve damage has not lasted sufficiently long to produce a *narrowing* of the mitral orifice.

Pericardial friction rubs may occur during the progress of acute endocarditis and are of value in showing that the inflammatory processes are still actively acute. A pericardial friction rub which may have existed before the onset of the acute endocarditis is of course not of the same significance.

Sufficient has been said upon the character of the auscultatory findings in acute endocarditis to impress upon the reader the necessity of making *frequent and repeated examinations of the heart*, at each visit to a patient who is suffering from acute rheumatic fever, from chorea or from the more severe and more highly toxic forms of tonsillar infection. Indeed, heart examination is a routine procedure not to be neglected in *any* acute infection.

DIAGNOSIS OF ACUTE ENDOCARDITIS.

The diagnosis is based, first, upon the history of a recent infection, particularly upon the history of acute rheumatic fever, chorea or tonsillar inflammation that has been unusually severe or protracted. Of the physical findings, the most significant is an alteration in the heart sounds, which alteration gradually pro-

gresses in sequence and eventuates in a systolic murmur located at the apex. Cardiac weakness is also progressive in acute endocarditis. The continuance of fever after an arthritis or other acute symptoms have disappeared is a valuable diagnostic point. Further evidence that the process is still active in the body would be the *recurrence* of joint pains, chorea, tonsillar inflammations or of the erythema which is often present in acute rheumatic fever or arthritides. There is significance, too, in a leukocytosis which persists after the arthritis, tonsillitis or chorea have disappeared. Emboli are not as frequent in acute endocarditis as in that malignant form where an acute inflammation is engrafted upon previously established valve damage.

SYMPTOMS OF MALIGNANT ENDOCARDITIS.

As a rule this gravely serious condition is precipitate in its onset. There are instances in which it may develop slowly as the result of a low-grade bacteremia which persists after an acute systemic disease has subsided. Malignant inflammation of the lining membrane may also be merely a part of a general pyemic invasion of many organs. Or it may arise as a result of metastasis from any original septic focus, in which event there may be a "typhoid" form or a "cerebral" form. The condition may sometimes resemble malaria or even a kidney autointoxication. So, the symptoms may be referable to any organ and the clinical picture not at all dominated by symptoms which are clearly referable to the heart.

Embolism.—Emboli are to be expected in malignant endocarditis. They may be the cause of a renal

infarct, and a gross hematuria be the consequence. The spleen is often enlarged and painful as the result of an embolus. There may be fever which is of the septic or pump handle type, delirium, paroxysmal chills followed by sweating, loss of weight and cutaneous conditions; but these manifestations, after all may be grouped as but part of the picture which is furnished by *septicemia with embolism*. Infective aneurism may also form in various portions of the body. Hemiplegia and aphasia may result from cerebral embolism as may also insomnia and hallucinations. Visceral emboli may cause sudden violent pains, diarrhea and other gastro-intestinal symptoms.

The cutaneous manifestations may take the form of erythema, urticaria or subcutaneous nodules. The latter are often called "rheumatic nodules" and Brenneman interprets their presence as nearly always meaning endocarditis.

Physical Signs.—Cardiac enlargement is more likely to occur in malignant than in acute endocarditis and may be confirmed by percussion. Auscultation reveals the same changes in heart sounds as were mentioned under *acute* endocarditis; but *multiple* murmurs are more frequent in the malignant form, and to this an aortic murmur may be added. Pericardial friction sounds or pleural friction rubs may arise if either of these structures are coincidentally affected.

DIAGNOSIS OF MALIGNANT ENDOCARDITIS.

The knowledge of a valvular lesion having existed prior to the present acute illness is of importance when considering the possibility of malignant endocarditis being present. Embolic features, such as

sudden swelling of the spleen, sudden hematuria, hemiplegia, coldness or numbness in legs or arms are among the most significant signs. A septic fever which arises during the course of an acute infection which does not usually have such extensive temperature fluctuations is a suspicious circumstance. The occurrence of purpuric and painful cutaneous nodules has much diagnostic importance attached to it by various observers. Progressive cardiac changes, such as an increase in the transverse diameter of the heart, alterations in heart sounds which eventuate in murmurs or the occurrence of an aortic murmur, are signs of much significance. Blood cultures may reveal a causative organism which may also be found in the urine.

Malignant endocarditis is sometimes confused with typhoid fever, but the differentiation can be made by the sequence of typhoid fever symptoms and signs at the beginning of the second week of the fever. Widal reactions are of little value in these days of anti-typhoid inoculation, for inoculations negate the test; blood cultures may reveal the typhoid bacillus. Malignant endocarditis may at times be confused with malaria but a blood examination for the plasmodium of this latter condition will establish the differential diagnosis.

PROGNOSES OF VARIOUS TYPES OF ENDOCARDITIS.

Acute Endocarditis.—Those patients who suffer from acute endocarditis which has its origin in acute rheumatic fever or chorea usually recover but a degree of valve damage often persists. Those cases in

which positive blood cultures are eventually found frequently die.

If the general nutrition of the patient can be maintained during the progress of the inflammation and until the infection has spent itself, the patient is likely to recover; but if in spite of all efforts the general nutrition of the patient becomes more and more depraved, the outlook is ominous. The appearance of embolic symptoms and of eruptions are very serious signs. It should be remembered that acute endocarditis may either terminate in or undergo transition into the malignant form at any time during the progress of the condition.

Acute malignant endocarditis usually terminates in death. The duration of the disease is variable but short, the patient rarely surviving over a month or six weeks. The amount of emaciation during the disease is excessive; one patient in six weeks dropped from her usual weight of 180 pounds to 90 pounds at the termination of her illness. If recovery from acute malignant endocarditis takes place it is never complete and death may be the happier termination of the disease.

Chronic malignant endocarditis, on the other hand, may eventuate in recovery after an illness which is rarely less than four months and may cover a period of a year or more. Here, too, permanent damage to the valves of the heart quite constantly results.

TREATMENT OF ENDOCARDITIS.

Absolute and *complete* physical rest is of more vital importance in endocarditis than is any other conceivable therapeutic measure. *Absolute* rest is of

more importance in endocarditis than in any other disease to which the flesh is heir.

The patient with either acute or malignant endocarditis should not be permitted to even turn himself in bed, so necessary is it to conserve every particle of physical effort and cardiac strength. When one recalls that an affected heart which makes 12 extra beats a minute makes 17,280 cycles a day more than are required in health, one can then appreciate the necessity of sparing the organ even such a demand as would be occasioned by the simple effort of extending the hands. The rise in pulse-rate which so often follows attempted effort on the part of the patient does not usually cease when the effort ceases. The increase in rate will continue for a considerable time afterward. Hence, attempted effort means more exhaustion; more exhaustion means less resistance of diseased cardiac tissue; and lessened resistance of cardiac tissue may mean cardiac failure and death.

It is not the part of wisdom for the physician to attempt to impress the *patient* with the necessity for absolute rest; to do so may alarm him unnecessarily and cause him an anxiety which he might well be spared. The knowledge that someone is at hand to carry out his every wish is often sufficient information for the sufferer. Instructions should be given to the *nurse* or attendant, thus sparing the patient the added burdens of anxiety, alarm or depression. Gentle restraint is a much better method of controlling the invalid than is the employment of either force or argument.

Attention should be directed to combating the underlying cause. *Focal infections* (page 224), if

demonstrated in a patient acutely ill with endocarditis, present a fine question for decision as to what period of the disease their correction can be more safely attempted.

Should acute rheumatic fever be the provocative infection, the salicylates should be employed in 10 to 30 grain doses at 3 or 4 hour intervals—always sufficiently diluted, and always combined with sodium bicarbonate. The sodium bicarbonate is added for the purpose of counteracting gastric irritation and in the hope of preventing the possible occurrence of salicylate-poisoning by thus rendering the urine alkaline.

Chorea should be treated by the administration of *liquor potassii arsenitis* (Fowler's solution), beginning with 3 minim doses well diluted in water, *t.i.d.*, *p.c.* The dose is increased one minim at each administration until the physiologic limits of arsenical tolerance become manifest by slight puffiness under the eyes, looseness of the bowels and griping. When these symptoms occur the drug should be withdrawn for a day or two, and its administration again begun in a daily decrease of dose until a minimum of 5 minims *t.i.d.* is reached and then increased as before.

The hygienic indications of elimination should be met as required. Gentle catharsis is secured by fluid extract of cascara sagrada in 10 to 30 minim doses, which meets the indication of peristaltic stimulation. The nurse should keep the skin in active condition by tepid baths, followed by witch hazel applications. Diet should be of the form most readily assimilable and be free from any substance which, by provoking indigestion or fermentation, might add one iota to the load of the heart. Heat may be employed to relieve

the sensory disturbances caused by an embolus; opiates may be required for pain. Insomnia, which may be the bitterest antagonist of much-desired rest, should be controlled by quiet surroundings, well ventilated room and by the employment of opiates *in sufficient dosage* to secure the result desired.

The question of sufficient dosage will have to be decided in each individual case. There is no way of telling how a given individual will react to opiates. To set an arbitrary limit on the amount of morphine to be used is to have the drug often fail of its purpose. *Rest* despite pain and *rest* despite insomnia is an absolute essential in endocarditis; if rest is best secured by morphine, it is better to err in having given a little too much of the hypnotic rather than to have the patient continuously wracked with pain or tossed about by insomnia through having given too little of the drug. Repeated doses are usually not so effective as is sufficient initial dose; but repeated doses may have to be used until one learns what constitutes *sufficient* dose for the individual patient. He who hesitates to continue the use of morphine when its continuance is warranted through the fear that a drug habit may be induced, can reassure himself with the fact that the morphine habit is very rarely acquired in several days of legitimate employment of the drug; the habit is engendered through use of morphine over a period of many weeks or months.

Vaccine Therapy.—Much has been expected of the use of autogenous vaccines in endocarditis of the more severe types. An autogenous vaccine is one which is derived from a laboratory culture of those particular varieties of germs which are present in a

given case, as determined by bacteriologic examination and by cultures from the blood. When used at all vaccines should be used early. To employ them as a last resort in malignant endocarditis is to deprive the patient of a possible benefit in his battle with this desperate condition. Unfortunately, there may be a delay of several days in securing the autogenous vaccine and during that interval a "stock" vaccine (one already prepared and marketed) may be employed until the more desirable autogenous vaccine is prepared. As to the efficacy of this form of treatment much doubt exists—a doubt which entitles the patient to the benefit occasionally reported from the employment of vaccine in the more severe types of endocarditis.

Digitalis.—It may be necessary to support a heart, which is threatened with failure, by the use of tincture of digitalis in 8 or 10 minim doses at 4 or 5 hour intervals. The drug is *not* to be used as a routine but only to meet the indications of progressive cardiac weakness or threatened circulatory failure. One of the foremost therapeutists of the day aptly refers to digitalis as "a lash to the lagging heart." To apply the lash to a heart which is already putting forth its supreme effort in combating its infection may result in the unloading of the last atom of reserve force of which that diseased heart is capable. Hence the thoughtless, routine employment of digitalis may precipitate death.

Convalescence.—The duration of the absolute rest period in those patients who have weathered the storms of acute or malignant endocarditis is a matter of much importance. There is no disease of

the heart which requires a longer convalescent period or a more careful guarding of the patient as he nears the shores of health. The absolute rest period should be continued until long after the fever has subsided and the rhythm of the pulse has become normal; only then should the patient be permitted to sit up for brief intervals. These intervals may be gradually lengthened as the days pass, provided no unfavorable change in the rate and rhythm of the pulse ensues. This usually means six or eight weeks in bed and another month on the couch before any activity is resumed. Even then, six months or more may elapse before the patient is permitted to busy himself in his former accustomed manner.

It may be observed during the convalescent period that the heart has acquired a change in rhythm, such as premature contractions, or auricular fibrillation, which may have set in during the course of the acute disease and from now on be permanently established. If such a permanent irregularity has occurred, it is not an indication for keeping the patient at absolute rest *so long as the pulse rate is not markedly altered* by postural change or by exercise.

Despite the efforts of the physician and despite whatever care he may take in treatment, acute inflammation of the valves of the heart is quite likely to result in structural alteration of the orifices. The more thoroughly the likelihood of such structural alteration is appreciated the more care will the conscientious physician exercise in order that he be in no way responsible for the permanent damage that would ensue in consequence of disregarding the absolutely essential element in treatment—REST.

CHAPTER XVII.

Chronic Valvular Disease of the Heart.

GENERAL CONSIDERATIONS.

CHRONIC valvular disease of the heart is a term which is applied to permanent structural alteration of the cardiac orifice leaflets.

Chronic valvular disease is of clinical significance only in so far as it is an expression of co-existing heart muscle involvement. It is *heart muscle failure* and not a "leaking valve" that gives the classical symptoms of dyspnea, cyanosis, visceral congestions, etc. The part which an affected valve alone might play in weakening cardiac muscle to such an extent, is a part that is certainly overshadowed by the cardiac infection which, in all probability, attacked both muscle and membrane simultaneously, damaging muscle as well as valve.

Therefore, to attempt to appraise a heart lesion by auscultatory findings alone, without obtaining information as to the condition of the heart muscle, is to render oneself liable to grave errors in judgment. History, inspection, palpation, percussion, mensuration and rate-response-to-exercise should all be considered in their relation to the auscultatory phenomena dwelt upon in this chapter, for only with such complete information before us can we hope to correctly appraise a heart. Only by a careful weighing of the evidence obtained by all of these methods of

diagnosis can the physician intelligently answer the question which is of paramount importance to the patient, namely; "Is this heart condition serious—does it necessitate a complete change in my manner of life?"

ETIOLOGY OF CHRONIC VALVULAR DISEASE.

As chronic valvular disease is so often the ultimate result of a previous acute endocarditis it is evident that the same causes are productive of both conditions. It is also likely that another cause of chronic valvular disease exists, namely, an insufficient rest period following acute affections of the heart. The value of rest in the prevention of chronic disease of the muscle and of the endocardium has been dwelt upon exhaustively in the chapters preceding, but its importance might well be here again emphasized by drawing attention to the fact that *an insufficient rest period* following the cardio-circulatory demands of *any* acute infection is a *most likely cause of permanent structural alteration of heart tissue*.

There is no particular cause for a particular valve lesion. While it is of course generally recognized that acute rheumatic fever affects, for the greater part, the mitral valve; and that syphilis, for the greater part, invades the aortic region and the aortic valve; yet either one of these infections may involve any valve or every valve of the heart. The broader concept of heart affections requires one to adopt the view that *any* infection, acute or chronic—whether it be local, focal or general—may be reflected in disturbances of the heart. The heart leaflets may also be damaged as a result of the same sclerotic changes in heart tissue which take place in the walls of the

arteries. Chemical poisons, defective elimination or autointoxication, when long continued, may damage the valves of the heart.

In considering the etiology of acute and malignant endocarditis it was stated that in those conditions the causative bacterial invasion was still active. When acute endocarditis terminates in chronic valvular disease the bacterial invasion has become quiescent; it is no longer active. It is of course possible for an acute infection to be engrafted on an old valvular lesion, but as a usual thing chronic valvular disease when once established is not actively progressive in nature. It may seem to increase in severity as the years go by, and become more and more incapacitating with the passing of time; but these are changes induced by the gradual wearing out of cardiac structure or by the eventual exhaustion of damaged muscle which has for years been working under load and strain.

MORBID ANATOMY.

Chronic valvular disease exhibits many varieties of pathologic change. There may be simple *thickening* and induration of the valve leaflets or the leaflets may be *retracted* or curled upon themselves; *atheroma* and *calcification* may render the valve totally unfit as a functioning structure. The term "funnel shaped mitral" describes an atheromatous valve which has been drawn down into the cavity of the ventricle by contractions and thickening of the chorda tendineæ and muscoli papillares, giving the valve the funnel shaped appearance from which it derives its name (Fig. 67). "The button hole mitral" is a term that was employed by Corrigan to describe adhesions and

atheroma of the mitral valve. Normally, the mitral valve will admit the tips of three fingers; in the "button hole mitral" the orifice is barely sufficient to permit the entrance of a button (Fig. 68).



FIG. 67.—THE FUNNEL-SHAPED MITRAL.

The mitral valve has been drawn into the cavity of the left ventricle. A glass rod passes through the stenosed mitral valve. The black arrow points to thickened and foreshortened tendinous chords, which certainly acted in holding the valve open throughout the cardiac cycle, causing insufficiency as well as stenosis of the orifice during life. (Jefferson Medical College Museum.)

VARIETIES OF VALVULAR DISEASE.

A valve may fail to close properly; in such a circumstance blood regurgitates through the imper-

fect opening when the heart contracts. The valve is *insufficient*. Or a valve may be shrunk, narrowed and thus obstructed as a result of disease. The word which is used to express narrowing is *stenosis*.

Any valve may be insufficient or it may be stenosed, or it may be both. There are many logicians who believe that stenosis cannot be present without insufficiency co-existing. Insufficiency is often followed by stenosis—a progressive step, as it were, from one condition to another. A variety of valve lesions may exist in the same heart, as shown in the following paragraph.

INCIDENCE OF VALVULAR DISEASES.

The left side of the heart is more often affected than is the right side. *Congenital* valvular defects, however, form an exception to this rule as they are in the great majority of cases right sided. The order of frequency of valve lesions in the left heart is: (1) mitral insufficiency; (2) mitral stenosis; (3) aortic insufficiency; (4) aortic stenosis. The relative incidence of right sided lesions, which are infrequently found and still less frequently diagnosed, are: (1) tricuspid insufficiency; (2) tricuspid stenosis; (3) pulmonary stenosis; (4) pulmonary insufficiency. It has been estimated that right sided lesions constitute a fraction less than one one-hundredth of the total number of cases. In combined lesions, double aortic and mitral insufficiency predominate, with aortic stenosis and mitral stenosis occupying second place: aortic and mitral insufficiency are third in frequency; while double aortic and double mitral lesions are the least frequent of all the combined valvular affections.



FIG. 68.—THE BUTTONHOLE MITRAL.

Near the center of the photograph is a depression which in a healthy heart is a valve opening which admits the tips of 3 fingers. The valve here shown is so stenosed that only the small, semilunar slit at the upper part of the depression remains open. (Jefferson Medical College Museum.)

Statistics have been gathered by Gillespie in reference to the percentage of valve lesions which he found. The mitral valve was affected in 58 per cent. of the cases, the aortic in 21 per cent. Mitral and aortic lesions co-existed in 19 per cent., while pulmonary and tricuspid lesions were found in only 0.8 per cent. of the group. More than $\frac{3}{4}$ of all valvular lesions in women occur at the mitral orifice. Aortic disease is three times as common in men as in women. Double lesions occur with twice the frequency in males.

MITRAL INSUFFICIENCY.

This is considered a very common valvular lesion and also the least harmful. My records show it to have been the most frequent diagnosis which was made in cases which were referred for murmurs, which murmurs were later found on extended study to be without significance (see page 78). In other words, the diagnosis had been based solely upon a systolic murmur, or upon some other isolated and unassociated sign, rather than upon *a definite group of symptoms and signs*. It is believed by some physicians that mitral insufficiency is diagnosed far more frequently than necropsy findings warrant. Such an opinion is probably true when one remembers that mitral insufficiency may be of the muscular type; it is quite possible for the atrio-ventricular orifice to fail to close on account of defective muscular action, and a murmur arise in consequence. There are also febrile conditions which induce transitory alterations in muscular tone; anemia, too, may interfere with cardiac nutrition; under such circumstances a mitral valve may fail to properly close and a murmur be

heard. Manifestly, a diagnosis of mitral insufficiency made under such conditions as these will not be revealed at necropsy; there is no way of estimating muscle-tone or of estimating the changes due to muscle-anemia at the post-mortem table. But to flatly assert that such a condition as mitral insufficiency cannot exist is to deny the evidence of contracted mitral leaflets, inversion of their edges, vegetations, calcareous plates, and shortness and degeneration of the tendinous cords which are so frequently seen at autopsy, and which must have rendered the valve as patulous and as insufficient during life as it was found to be after death.

The diagnosis of mitral insufficiency is based on the following *group* of physical signs: (1) an apical systolic murmur; (2) its transmission to the left axilla or even to the angle of the scapula in the back (this latter region sometimes being called the "mitral area" for the reason that a mitral systolic murmur can there be heard); (3) accentuation of the pulmonary second sound; (4) increase in the transverse diameter of the heart. To these physical signs are to be added in varying degree the symptoms of cyanosis, shortness of breath, edema, limited response to effort, etc. These latter symptoms are not characteristic of mitral disease; they are not characteristic of any valvular disease; they are evidence of *heart muscle involvement*.

MITRAL STENOSIS.

Mitral stenosis should be carefully studied. It is not a diagnosis which readily lends itself to snap judgment. Necropsies often fail to confirm the diag-

nosis of mitral valve narrowing, for the reason that it is frequently confused with the overacting heart of emotional persons or neurasthenic individuals. The diagnosis should be based upon the presence of the following group of physical signs.

(1) An apical presystolic murmur, sharply localized at that point on the chest wall which corresponds to the clinical apex of the particular heart being examined; (2) an apical presystolic thrill; (3) a "snappy" first sound as heard at the apex; (4) an accented pulmonic second sound; (5) an increase in the right diameter of the heart. To these are frequently added the following associated physical signs: (A) an enlarged left auricle which is often present in this condition and which may press on the recurrent laryngeal nerve, thus producing a husky voice or a "brassy" cough. (B) Pulmonary congestion may induce a bloody sputum which is more often encountered in mitral stenosis than in any other valvular disease. This is the lesion in which the "heart failure cells" which are referred to under myocarditis (page 203) are often found. (C) Jugular pulsations are a very frequent part of the clinical picture.

(D) *Auricular fibrillation* was accompanied by mitral stenosis in 52 per cent. of a group of cases which Lewis studied. Hence the characteristic pulse changes of auricular fibrillation are noted more in this than in any other chronic valvular disease. Heretofore the name "pulse irregularis perpetuus" was applied to the pulse which older writers found so often in mitral stenosis. This pulse is recognized by the fact that it is continuously, persistently and absolutely irregular as to rate, as to rhythm and as to

volume; and by the further fact that the irregularity in all three of these respects increases when the heart-rate is increased by effort. (See Auricular fibrillation, page 253.)

“Clubbing of the finger tips” is a time-worn sign of mitral stenosis, believed to be present when the lesion is of long standing; the sign is mentioned here to accord it the respect which should be paid its age, —not because it is of clinical significance or of diagnostic value in heart affections.

Discussion of Essential Physical Signs in Mitral Stenosis.

In order to understand the phenomena of mitral obstruction, it is necessary to apply some of the statements which were made under the physiology of the heart. One should first remember that in a heart beating at the rate of 75 contractions a minute, systole occupies 0.5 of a second and diastole occupies 0.3 of a second; in the last 0.1 of a second of diastole, auricular contraction occurs. Secondly; the first sound of the heart is produced by ventricular muscular action and by the tautening of the mitral and tricuspid valve curtains.

The *duration of the murmur* in mitral narrowing is probably dependent upon the degree of the valve damage. Early in the condition the murmur may be very short and be heard only during that period when the auricle is actively contracting, *viz:* at the end of the rest period of the heart. It is in the last 0.1 of a second of diastole that the auricle contracts. Hence the presystolic murmur in early cases may be only 0.1 of a second in duration. As the narrowing and

shrinking of the leaflets progresses to a degree where they exert no restraining influence whatever on the blood which is accumulating in the auricle previous to contraction of that chamber, the murmur may occupy *all* of the diastolic period—perhaps the full $\frac{5}{10}$ of a second which diastole occupies in a heart contracting at the rate of 75 beats a minute. Under such a circumstance the *crescendo quality* of the murmur is more apparent,—low and rumbling it may be at first, then increasing in intensity as the inception of auricular contraction forces blood through the narrowed orifice with greater impetus than that which was given the fluid by gravity and by the “aspirating” action of the ventricle. Instantly the first sound of the heart occurs: it is likely that the ventricle attempts to overcome the valve defect by increased muscular action; this in turn increases the closure-force of the tricuspid, the fellow-valve of the opposite side; both factors produce the “snappy” first sound.

The Effect of Auricular Fibrillation.—The presystolic murmur of mitral stenosis undergoes marked alteration in the presence of auricular fibrillation. In this condition the auricle stands in trembling diastole. There is no auricular contraction. Hence there is no presystolic murmur. However, an *early diastolic murmur* due to ventricular filling, may be heard if the heart-rate be deliberate.

An apical presystolic murmur is essential if one is to make a diagnosis of mitral stenosis. It is *not the only sign which is necessary* for the diagnosis, but it is the prime requisite to which the *other signs should be attached*. The presystolic murmur should be constantly present; it is louder when the patient lies on his

left side, and while it may thus alter in intensity, it should be so definitely present at repeated examinations as to cause neither doubt nor vacillation in the mind of the examiner. Moderate exercise may intensify the murmur: violent exertion may obscure it completely, for the reason that violent exertion produces increased heart-rate; increase in heart-rate is quite invariably at the expense of and shortens diastole; and the shortening of diastole by a marked increase in rate may blur out the presystolic—really a late diastolic—murmur.

A *Flint murmur*, first described by the elder Flint whose name it bears, may be confused with the murmur of mitral stenosis. This murmur, also, is presystolic in time and is heard at that point on the chest wall which corresponds to the clinical apex of the heart; it may also be accompanied by a thrill: hence the confusion. The Flint murmur, however, *arises only in aortic insufficiency*. It is produced by the anterior cusp of the mitral valve interposing itself from its usual position along the heart wall during diastole, into the ventricular blood-stream; thus the leaflet is made to vibrate, first, by the blood which falls back from the imperfect aortic valve; second, by the blood which passes through the orifice during ventricular filling. Hence there is a late diastolic (which is of course a *pre-systolic*) murmur and thrill at the apex. The differential diagnosis is made by searching for the Corrigan pulse, arterial throbbing, increased transverse diameter of the heart and other phenomena of aortic insufficiency.

The Presystolic Thrill.—This thrill may not be present in early cases where the murmur is slight;

the same factor that produces a murmur produces a thrill, *viz*:—a narrowed orifice. If the opening be only a little narrowed but little thrill ensues. It is very easy to confuse the systolic vibration of an over-acting heart such as is often seen in the cardiac neuroses, with this thrill.

The Right Transverse Diameter of the Heart. The right border of the heart is formed by the right auricle; the auricle is not usually enlarged to percussion in the early stages of mitral stenosis, but later the auricle may attain a size equal to all the other chambers of the heart. In such an event the right border of the heart, as measured in the 4th inter-space, will be much greater than the customary distance of 3 cm. from the midsternal line. One would probably be correct in assuming that marked increase in the right border of the heart in mitral stenosis is a sign that the stenosis is well advanced.

Evidence of mitral insufficiency frequently co-exists with mitral stenosis, such evidence being afforded by a distinct, harsh and blowing apical *systolic* murmur, transmitted to the left. To be of significance this systolic murmur should be attended with increase in the *left* transverse diameter of the heart.

AORTIC INSUFFICIENCY.

The astute Irish physician Dominic Corrigan, (1802-1880) writing in 1832, first described this condition in a masterly manner which has caused it to be since referred to as "Corrigan's disease." He spoke of it as "a permanent patency of the mouth of the aorta."

This is that form of valvular disease so often found at necropsy in cases of sudden death occurring in those who are active and apparently robust. The incidence of syphilis in cases of aortic insufficiency

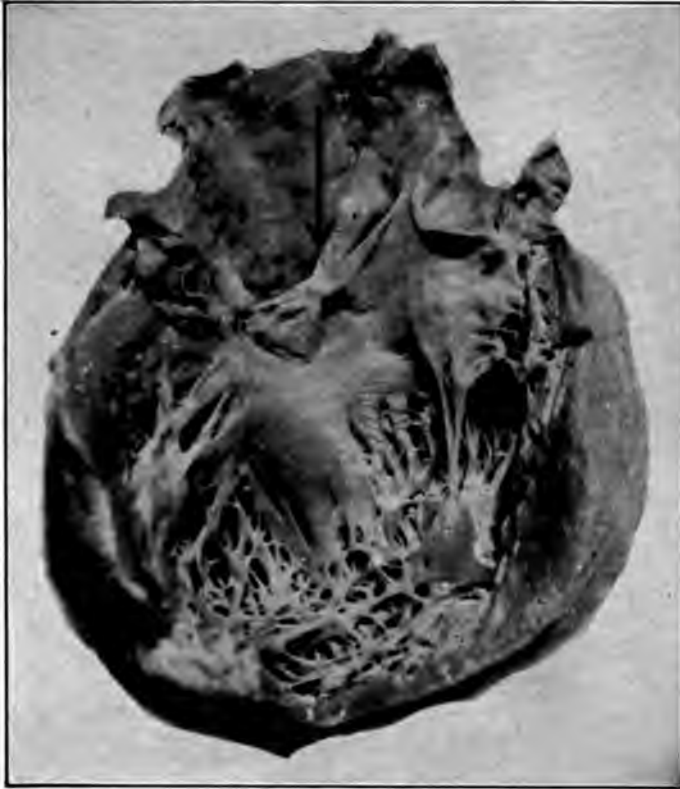


FIG. 69.—FUSION OF AORTIC LEAFLETS.

(University of Pennsylvania Medical School Museum, collection of *Dr. R. S. Willson*.)

has caused many writers to classify this valvular disease under the caption of cardiovascular syphilis. The frequency with which syphilis invades the aorta was shown in the statistics of Warthin of Ann Arbor,

who was able to demonstrate the *Spirocheta pallida* in the aorta of virtually 80 per cent. of successive and unselected autopsies. This is proof sufficient that syphilis may be a large factor in producing inflammation of the aorta and of the contiguous aortic valves, but to assume that syphilis is the *only* etiologic factor in the production of aortic valve involvement is to deny a mass of clinical evidence which shows that there are many non-venereal factors which enter into its production.

A lesion of the aortic valve may be a heritage from endocarditis which has been produced by any bacterial invasion. The valve may also be insufficient because it is congenitally malformed. It may be sclerosed—and be the only valve thus affected—by the processes which produce atheroma of the arteries in arteriosclerosis.

Lesions of the aortic valve are not as a general thing the result of an active, fulminating infection. They are more the result of a change which is of slow development (Fig. 69), such as would be brought about by a long continued systemic infection or by atheromatous changes in and about the valve, as in cardiosclerosis.

Physical Signs: Inspection.—The appearance of the patient with aortic insufficiency is usually striking. Arterial throbbing which is systolic in time may be seen in the temporal arteries or in the vessels of the neck. The head may nod with each pulsation of the heart. The precordial impulse is diffuse and forceful. The heart, even upon inspection, is manifestly enlarged downward and obliquely toward the left. Despite the fact that the precordial impulse is often

diffuse one may at times be able to locate *two* impulses at the apex of the heart. This is due to the fact that the "*clinical*" apical impulse is caused by contraction of the right ventricle; the "*anatomical*" apex of the heart is at the tip of the left ventricle: In enlargement of the left ventricle that chamber may be so distended that the anatomical apex comes in contact with the chest wall and presents the second impulse.

Quincke described a capillary pulse in aortic insufficiency which can be elicited by making gentle pressure on the end of the patient's finger nail, or it may be better seen if a microscope slide or other convenient piece of glass be placed upon the lips. The color deepens with each pulsation of the heart and fades between beats. The phenomenon is of little value as a diagnostic point in aortic insufficiency, for it is seen as well in exophthalmic goiter, in occasional forms of anemia, in neurasthenia and in other conditions of vasomotor instability. Dermographia, a condition in which designs can be traced with a blunt instrument or with the finger nail upon the flesh of the patient, and the design promptly be outlined in red and raised upon the surface of the skin, is frequently present in aortic insufficiency. Dermographia, however, is of no diagnostic importance.

Aortic insufficiency is more frequently accompanied by a pallor which is suggestive of anemia than is any other valve lesion. This valve lesion also frequently co-exists with angina pectoris, probably because syphilis, an infection which is a frequent cause of the one condition quite as frequently causes the other. Syphilitic invasion is also a very probable explanation for the pallor which accompanies many

cases of aortic insufficiency: this would indeed seem to be the logical explanation of the pallor when one recalls cases of aortic insufficiency in children, where there is no evidence whatever of syphilis, and in whom there is usually a florid face and a heightened natural color.

Palpation.—By palpation one may confirm the heavy tumultuous impulse and note the presence of systolic shock at the precordium. There is an increase in the transverse diameter of the heart, an increase which may be so marked as to suggest a colloquial term by which aortic insufficiency is known, namely “ox heart.”

The pulse is perhaps the most characteristic and most constant phenomenon of aortic insufficiency. It is a jerky pulse of full expansion; the waves rise suddenly and with extraordinary force, then instantly collapse, the artery seeming to be quite empty of blood between beats. Various names have been used to describe this pulse. It is the “*pulsus celerrimus*” of the older writers; it is the “water hammer pulse” of more recent authors, it is the “pistol shot pulse” of others, and it also bears the name of Corrigan, the one who first described it. The collapsing character of the *pulsus celerrimus* is intensified if the radial artery be felt when the wrist is held over the patient’s head. Its characteristics may be masked by arteriosclerosis.

Percussion.—Percussion will confirm the downward and outward increase in the left border of the heart. It may when practised in the second interspace to the right and to the left of the sternum, show an increase over the usual distance of $5\frac{1}{2}$ cm. which

demarks the usual extent of dullness of the aortic arch in men, and thus indicate dilatation of the transverse arch of the aorta—which is frequently associated with aortic valve lesions.

Auscultation.—There is a basal diastolic murmur which is faint, prolonged, blowing and which has a *diminuendo* characteristic. The *diminuendo* of the murmur is in contrast to the *crescendo* of the bruit of mitral stenosis, in which latter condition the sound *increases* in intensity from its beginning; but the diastolic murmur of aortic insufficiency *decreases* in intensity from its beginning. The murmur is best heard with the patient in the sitting posture, the body bent a trifle forward. Classically, the murmur should be heard at the aortic punctum maximum—in the 2d interspace to the right of the sternum—but as a matter of clinical fact, it is indeed oftener heard at the pulmonic area in the 2d interspace to the *left* of the sternum; even lower, in the 3d and 4th interspaces to the left of the sternum, it may attain its greatest intensity. When this murmur is listened for with the stethoscope it often escapes detection; its faint and high pitched note can be best appreciated when the ear is laid directly upon the chest. It begins just after the second sound of the heart at the aortic area and may muffle, prolong, or even replace the second aortic sound.

The first heart sound at the base is often obscured by a *systolic bruit* which may be caused by (1) roughened aorta leaflets, (2) by atheroma, (3) by dilatation of the aorta, or (4) by stenosis of the aortic orifice. Thus we have *two* murmurs,—the “to and fro” basal murmur of double aortic lesions—which is

by *no* means always produced by the co-existence of aortic insufficiency and aortic *stenosis*.

The *Flint* murmur (see page 253) is frequently audible at the apex. Its presence was noted in 57 per cent. of my records of aortic insufficiency.

Arterial Sounds: The "Pistol-shot Femoral."—The normal femoral artery is devoid of sound. In aortic insufficiency, auscultation over the femoral artery may reveal the presence of sound therein, to which phenomenon the term "pistol-shot femoral" has been applied. The term is a poorly chosen one—it suggests a volume of noise and it suggests the element of surprise, neither of which is marked except in advanced cases. The sound varies in intensity—it more often resembles the click of a misfire, rather than a shot from a pistol.

Duroziez described a sound which may be present in aortic insufficiency. It is evoked by making slight pressure over an artery with the bell of a stethoscope. While heavy pressure may narrow the lumen of any artery and thus cause the production of sound, the phenomenon to which *Duroziez* drew attention is the *second* murmur, which occurs during collapse of the artery. It is this second murmur then, which is due to arterial collapse, and not the first murmur which occurs during filling of the vessels, which is essential to the establishment of *Duroziez's* sound. The phenomenon is not at all a constant one and is more frequently absent than present.

Blood-Pressure Estimates.—Blood-pressure estimates should be made in every suspected instance of aortic insufficiency. They may yield two important bits of testimony. The first point is one to which

Leonard Hill drew attention and is evolved by applying the cuff of the blood-pressure apparatus to the leg of the recumbent patient. It is found by this maneuver that the systolic pressure in the femoral artery may be 30 or 60 millimeters higher than it is in the brachial; indeed it may transcend these figures to a surprising degree. It is essential that the patient be in the recumbent position if Hill's sign is to be of any value; a difference between the brachial and femoral pressure naturally exists in a healthy person should the cuff be applied to the leg when he is *standing* or *sitting*.

The second observation which may be made during blood-pressure estimates is noted on auscultation of the brachial artery below the compression cuff. This maneuver shows in many instances, but not in all, that the loud systolic rap does not disappear at the usual and expected point; the sound persists as the needle approaches zero on the dial. Both Hill's sound and the one just mentioned are more frequently present than absent.

As to the systolic pressure in aortic insufficiency; while it is usually elevated, it may quite as often show no degree of elevation over what would be expected for the age of the patient. The pulse pressure, however, is quite constantly high.

Diagnosis.—The group of five cardinal points upon which the diagnosis of aortic insufficiency should be based are: (1) A basal diastolic murmur, best heard when the patient is sitting and bending a trifle forward; (2) an increase to the left of the transverse diameter of the heart; (3) a characteristic jerky pulse of full expansion which is followed by a sudden col-

lapse; (4) the presence of arterial sounds; (5) arterial throbbing.

"Heart Disease Delirium."—This term has been applied to the mental symptoms which frequently are found in persons who are suffering from aortic lesions. The symptoms consist of insomnia, melancholia, suicidal mania and delirium. Manifestly their occurrence is simply a coincidence and they are in no wise a result of any lesion of the aortic valve. It is highly probable that if syphilis be the responsible factor for an aortic lesion, it can also be the responsible factor for changes in the mental condition and in the nerve stability of the same patient.

AORTIC STENOSIS.

Aortic stenosis is the rarest uncomplicated valve lesion of the left heart. In 250 necropsies at the Massachusetts General Hospital there was not one of uncomplicated aortic stenosis. It nearly always co-exists with insufficiency of the aortic valve. It is a process of slow development and the leaflets do not present the changes which one sees in tissue as a result of frank inflammation. Aortic stenosis is more often seen in those of advanced years, although there are rare instances where it may be congenital.

Inspection and palpation reveal a forcible impulse which is produced by the enlarged heart. This impulse which is a slow, deliberate push, is characteristic of aortic stenosis. Palpation of the pulse confirms the deliberate ventricular action; the pulse in narrowing of the aortic valve is small and late and has a sustained plateau—in contradistinction to the quickly collapsing pulse of aortic insufficiency. To

this pulse the name *pulsus parvis et tardus* is given by the older writers.

Percussion shows an increase in the left transverse diameter of the heart. The increase is not so pronounced as is that of aortic insufficiency. Auscultation reveals a basal systolic murmur at the aortic area which is conducted into the carotids. The murmur is usually harsh and loud but in exceptional instances may be soft and musical. While it is generally best heard at the aortic punctum maximum, there are instances in which it may be heard at the left border of the vertebral column at about the level of the fourth dorsal vertebra. This is the point at which the aorta first comes close to the spine. A basal systolic thrill rougher than that which occurs in mitral stenosis is present and is conducted to the vessels of the neck. The second sound of the heart as heard at the aortic area is weakened. The reason for this is clear when one recalls that the second sound of the heart is produced by the closing of the aortic and pulmonary valves; if the aortic valve be stenosed, manifestly it cannot close, and that part of the second sound which is due to the normal action of this valve is absent; hence the second sound is feeble.

Diagnosis.—Basal systolic murmurs frequently occur and may be caused by intimal roughening of the aorta or by change in the caliber of the vessels, no matter whether that change be produced by dilatation of the aorta, by atheroma, by narrowing of the lumen or by any other factor which would alter its caliber. Hence a basal systolic murmur at the aortic area, if one is to interpret it as a sign of aortic stenosis, must have as its accompaniment a basal systolic

thrill, weakening of the second sound of the heart at the aortic area, and the slow, deliberate well sustained pulse which is characteristic of the valvular lesion under discussion.

TRICUSPID INSUFFICIENCY.

As with all valvular diseases of the right heart, tricuspid insufficiency is of rare occurrence and yet it is of greater comparative frequency than is insufficiency of the pulmonary valves. This observation applies only to the tricuspid insufficiency of structural change for insufficiency may also be due to a muscular cause which occurs when the right ventricle is relaxed or enlarged. *Muscular* insufficiency of this valve is of *frequent* occurrence, being found in acute conditions of the lung which obstruct the lesser circulation, or associated with such chronic pulmonary diseases as emphysema or fibroid phthisis. When pulmonary conditions throw a load on the right ventricle the caliber of that chamber increases, and the valve leaflets stretch in response to the muscular pull. This is the traditional "safety valve regurgitation" of the tricuspid valve.

The muscular "*safety valve*" type of tricuspid incompetency may afford no physical signs, usually none other than a soft systolic blow. *Structural* lesions of this valve, on the other hand, may produce marked symptoms. There is often edema, first of the feet, then of the ankles, and then ascites; there is distension of the jugular vein; by pressing a length of this vein between the fingers one will notice, upon releasing the lower finger, that the vein fills from *below*. Both this sign and visible pulsation of the jugular

may be more noticeable on the right than on the left side. There is cyanosis, pulmonary distress, dyspnea and cough.

Physical Signs.—There is usually pulsation at the lower end of the sternum. The apical impulse is not displaced. A thrill is more often absent than present. The transverse diameter of the heart is sometimes increased to the right. The liver may be definitely pulsating; hepatic pulsation should not be confused with the impact which an enlarged heart might convey to the liver, nor should it be confused with abdominal movements. The edge of the liver may extend considerably below the level of the ribs.

Auscultation.—There is a soft, blowing systolic murmur best heard at the tricuspid area or at the xiphoid cartilage, which takes the place of the first sound of the heart at these areas. The murmur is conducted in the direction of the apex of the heart. The pulmonic second sound is faint.

TRICUSPID STENOSIS.

The rarity of tricuspid stenosis may be shown by the statistics of Herrick who was able to collect only 154 cases from the medical literature of the world. Of these 154 cases 90 per cent. were combined with mitral stenosis and only 12 times did tricuspid stenosis exist alone.

Tricuspid stenosis may be masked by the co-existing mitral lesion. Indeed it almost invariably exists in conjunction with a lesion of the mitral valve. Broadbent believes that tricuspid stenosis may be assumed when mitral stenosis, contrary to its usual rule, develops pronounced anasarca.

The physical signs consist of cyanosis, turgescient veins of the skin and dropsy. There is a short, rough, presystolic murmur at the xiphoid cartilage; the murmur is not transmitted and increases in intensity from its beginning. A rough presystolic thrill is present.

PULMONARY STENOSIS.

This condition is usually congenital. Of a group of cases which were studied, 96 per cent. were believed to have been the result of endocarditis during intra-uterine life. When the condition is acquired during adult life there is present a basal systolic murmur, which is loud and harsh and which replaces the first sound at the pulmonic area. This murmur is transmitted to the left clavicle but not into the vessels of the neck. The pulmonic second sound is feeble. There may be increase in the right transverse diameter of the heart. In the congenital type the same physical signs are present in a modified degree, and in addition there is more cyanosis, more urgent dyspnea and more marked venous turgescence than in the acquired type. A basal systolic thrill is present.

Simulating Sounds.

Systolic murmurs at the base of the heart, particularly to the left of the sternum, may be caused by several conditions with which we are familiar, and they may owe their origin to other circumstances with which we are not yet acquainted; their presence gives rise to erroneous diagnoses of pulmonary stenosis, unless one demands *associated physical signs* of the systolic murmur.

Balfour has called this area the "region of cardiac romance." Here may be heard cardiorespiratory murmurs, or the "hemic" murmur which accompanies anemia, or murmurs due to atheroma or to aneurism of the arch. In this situation may also be heard a murmur which may be produced when the edge of the left lung is retracted, thus permitting the conus arteriosus of the right ventricle to come in contact with the chest wall. Under such circumstances a murmur may arise due to pressure of the chest wall on this part of the right ventricle; and it may at times be intensified when pressure is made with the stethoscope, the patient being in the erect position.

PULMONARY INSUFFICIENCY.

As a structural defect this is the rarest of uncomplicated valvular lesions. It is the result of acute endocarditis which is secondary to acute infections. Congenital instances of the lesion are found at necropsy to be due to the fusion of two valve leaflets.

Cough, dyspnea and cyanosis are the logical sequence of pulmonary valve leakage. A soft thrill, diastolic in time, may be felt at the base of the heart. The right transverse cardiac diameter is traditionally increased. A basal diastolic murmur, soft and blowing in character, replaces or follows the second sound of the heart at the pulmonic area. The second pulmonic sound is feeble or absent.

Basal diastolic murmurs of aortic valve origin need cause little confusion when it is remembered that those due to *aortic* insufficiency are accompanied by the other confirmatory signs so diagnostic of Corrigan's disease.

CONCLUSIONS.

In the final analysis, to definitely name the valve affected and to definitely time the particular abnormal sound which occurs within the heart is simply to tie a *diagnostic tag* to the auscultatory phenomena incident to a heart lesion. It is of far more practical clinical importance to know *to what degree the heart muscle has been involved in the process which diseased the valve*. Murmurs do not tell us this. This information we obtain from the history, from the response of the heart to exercise, from changes in its rate upon moderate exertion, and from such physical signs as cardiac enlargement, pulse irregularities, etc. History, inspection, palpation, percussion, mensuration and rate-response to exercise, point far more definitely than do the revelations of a stethoscope, to the *ultimate purpose* of clinical cardiac diagnosis *viz: intelligent, corrective, constructive treatment*.

TREATMENT OF CHRONIC VALVULAR LESIONS.

There is no direct treatment for chronic valvular lesions. The disabling symptoms which arise in valvular disease of the heart are the result of myocardial insufficiency and not the result of local pathology in a valve. Valve lesions may improve to some degree as the condition of the heart muscle improves under treatment. Therefore the treatment of valvular disease is the same treatment as that for chronic myocardial change (Chapter XV). The treatment of chronic valvular disease which was once in vogue and which was known as the method of Oertel, consisted

in a series of graded exercises which increased the nutrition and muscular tone of the heart by judiciously combining periods of rest with frequent short excursions in hill climbing. The method is the equivalent of that suggested in Chapter XXIII under the caption of graded exercises.

.

CHAPTER XVIII.

Congenital Heart Affections.

CONGENITAL heart affections are of interest more from a pathologic than from a clinical standpoint, for the greater number of infants who are born with frank evidences of circulatory defects rarely survive. Evidence of congenital heart affections is rare in adult life.

VARIETIES.

Developmental malformations may be due to: (1) openings in the interauricular or (2) interventricular septum; (3) to a patent ductus arteriosus; (4) to malformations of the valves or deformities of the valve orifice. There may be (5) congenital malposition of the heart, such as dextrocardia. And there may exist (6) congenital valvular disease.

(1) *Defects of the Interauricular Septum.*—The foramen ovale may persist as an opening in the interauricular septum, or may continue into adult life only partially closed (Fig. 70). The foramen ovale is essential to the circulation of the fetus and is believed to completely close at birth. When it remains persistently open the phenomenon of “blue babies” is seen, probably due to admixture of venous with arterial blood in the greater circulation. There are those who contend that the cyanosis is produced by congestion incident to the abnormal opening, albeit evidence of congestion other than cyanosis is usually lacking.

Semi-patulous foramen ovale is more frequent in adults than is generally believed. Post-mortem studies which W. F. R. Phillips,¹ of Charleston, has conducted in this direction are of such interest that no better presentation of the subject can be made than

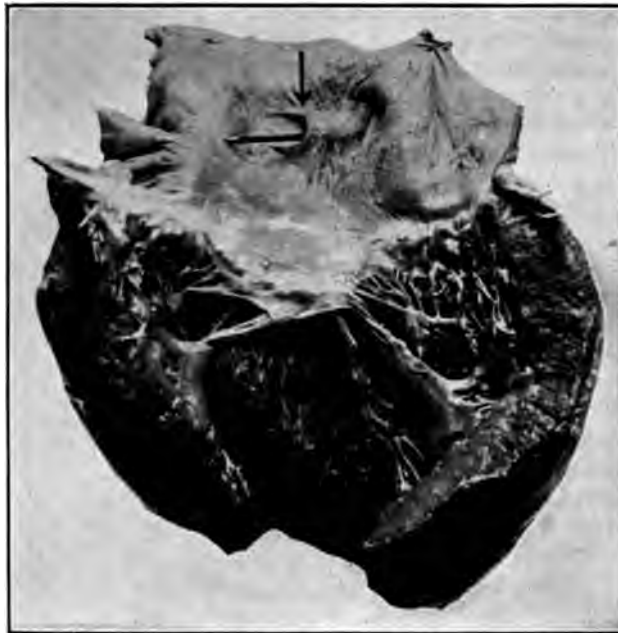


FIG. 70.—PATULOUS FORAMEN OVALE.
(University of Pennsylvania Medical School Museum, collection of *Dr. R. S. Willson*.)

to abstract a monograph² by Phillips, as is done in the following paragraphs.

The fact that such patulency was frequent has been remarked by anatomists and by pathologists

¹ Professor of Anatomy, Medical College of the State of South Carolina.

² Phillips: "Patent Foramen Ovale and its Relation to Certain Cardiac Murmurs;" *Medical Record*, Sept. 7, 1918.

from time to time with more or less emphasis; it has been commented on also by clinicians, but with less frequency and decidedly less emphasis than the prevalence of the condition deserves, at least such is the impression given by an examination of some of the current text and reference works. The series of hearts here reported (by Phillips) shows a ratio of persistency as against complete closure of the foramen ovale of 18 to 5, or 78 per cent. persistency to 22 per cent. closure. This is a ratio greater than any other so far within my knowledge. There appears to be wide variability in the observed frequency of unclosed foramen ovale in adult life. Quoting but a few among the relatively recent writers referring to the subject: Cunningham¹ gives the ratio of patency to closure as about 1 to 5, that is about 20 per cent.; Parsons and Keith² as over 26 per cent., as determined from a collective investigation of 399 hearts; according to Welch and Rolleston,³ Firket reported the foramen unclosed in 34 per cent. of his cases; Kolb and Wallman found the foramen persisting in no less than 44 per cent.

In length the passageways which Phillips observed varied from oblique slits to channels of some length, several being about three-quarters of an inch from orifice to orifice. Some of the long channels showed *culs-de-sac* extending from them. The caliber of the communicating openings varied from pin-hole to pencil-size or larger in diameter, one being large enough to admit the passage of the tip of the little

¹ Cunningham's Text-book of Anatomy, 4th ed., 1913, p. 875.

² Parsons and Keith: Jour. Anat. and Phys., London, 1897-8, p. 164.

³ Welch and Rolleston: Allbutt and Rolleston's System of Medicine, vol. vi, p. 762.

finger. The shape of the openings on the right was equally as variable as the locations; some were mere slits with slightly rounded margins, others dimple-like to funnel-like depressions, and others of irregular shapes.

The frequency of the occurrence of these openings through the fossa ovalis in adults of all ages and their almost invariable valvular nature, indicates that they are not inconsistent with perfect cardiac function, and that so far as constituting a pathologic condition they are virtually, as Humphry designates them, "without clinical significance." However, as factors that may and do cause unquestionably now and then signs that may be mistaken for those of pathologic changes in the heart, they are worthy of consideration.

They invest the significance of cardiac murmurs and their accurate determination and discrimination with a great degree of importance and responsibility. In the light of the probability of openings of the kind herein described, cardiac murmurs, especially systolic ones, unaccompanied by other corroborative evidence of functional impairment, become less indicative of real cardiac incapacity. How many healthy hearts have been doomed because of murmurs caused by open foramen ovals no one can say; one may guess. Apart, however, from any relation to cardiac murmurs, the frequency of openings in the fossa ovalis has a clinical importance not lightly to be passed by, because these openings are potential avenues for the passage of emboli from the venous circulation directly into the arterial circulation, the "paradoxical embolism" of Zahn."¹

¹ Phillips: *Loc. cit.*

(2) *Perforate Interventricular Septum*.—At no time in life is it natural for an opening to exist in this structure. It is a pure developmental malformation. It produces an intense cyanosis and the "*bruit de Roger*," which is a loud, harsh systolic murmur at the xiphoid cartilage, beginning early in systole and lasting through diastole. It is as amazing a sound as can come through a stethoscope, and is well compared with the grinding, scraping noise produced by drawing a knife over a whetstone. One instinctively feels that such a sound cannot originate at a valve; it must come from some unnatural communication between the chambers of the heart.

(3) *Persistent Ductus Arteriosus* (The ductus Botalli).—In the fetal circulation the ductus arteriosus is a communication between the left branch of the pulmonary artery and the aorta. It enables blood from the upper portion of the fetus to reach the placenta by way of the aorta. This vessel should close completely a few days after birth; it then becomes the ligamentum arteriosum. However, if the pulmonary artery be stenosed or if the aortic opening be narrowed, the ductus arteriosus may persist, thus becoming a congenital malformation. Under these circumstances it produces a loud and harsh basal systolic murmur which lasts into diastole. It is best heard at the second interspace. It has a diminuendo characteristic. This murmur may also be heard over the scapula. A long, rough thrill may be felt over the base of the heart.

(4) *Valve Defects*.—The heart valves are subject to a variety of structural changes. There may be more leaflets in a valve than is normal, or there may be



FIG. 71.—IMPERFECT AORTIC VALVE.

Two leaflets of the aortic valve, instead of the customary three are not incompatible with longevity and are detected only at necropsy. One of the valves here shown is the seat of a healed ulcerative defect. (University of Pennsylvania Medical School Museum, collection of *Dr. R. S. Willson.*)

less (Fig. 71). The valve leaflets may be perforated. Occasionally there may be no attempt whatever made at the formation of leaflets, and the valve be a mere diaphragm which stretches over the natural opening of the heart. Congenital valve defects may also assume the shape of a funnel. Defective or malformed valves are the easy prey of endocardial inflammations.

(5) *Dextrocardia*.—Dextrocardia means complete transposition of the heart from the left to the right side of the chest. It is usually associated with a transposition of the abdominal viscera and is usually discovered accidentally, although attention may be drawn to it by cardiac symptoms which arise when *general* visceral transposition is not complete, as when the heart is on the right side and subject to pressure-symptoms from the liver below.

Dextrocardia may not be complete; the heart may be in neither right nor left chest, but occupy a medial position. Under such circumstances a distinction must be made between *transposition* and *malposition*, for the heart may be misplaced to the right as the result of a left sided pleural effusion which pushes it into an unnatural situation (see Figs. 11, 12 and 13); or a fibroid right lung may, in its shrinking, so retract the mediastinum that the heart is pulled toward the right.

(6) *Congenital Valvular Disease*.—Congenital valvular disease is confined, for the most part, to the right side of the heart. Of the lesions which may occur on the right side, 69 per cent. of them are caused by pulmonary stenosis. It is the result of endocarditis during uterine life. Cyanosis is the most striking and the most common evidence of congenital

pulmonary stenosis. The physical signs in the congenital form are the same as those which arise during extra-uterine life. There is a harsh, basal systolic murmur which is transmitted to the clavicle; there is a thrill at the base of the heart; the pulmonic second sound is weak; and there is an increase in the right transverse diameter of the heart.

Congenital pulmonary stenosis is usually combined with an opening in the ventricular septum; hence the diagnosis of this valvular disease is not always possible, for multiple signs due to developmental defects may overshadow or confuse the picture.

CONCLUSION.

The six abnormalities briefly discussed herein by no means constitute the full extent of malformations that may occur in the heart; absence of the pulmonary artery or an extra pulmonary artery or even absence of one or the other of the heart chambers are examples of the infinite variety of developmental defects which are found post-mortem. The six varieties enumerated, however, constitute the more frequent defects—comparatively speaking—which live long enough to present problems in physical diagnosis.

Even though one establish such a diagnosis to one's satisfaction, there yet remains the problem of treating a patient who has a congenital heart affection—a problem upon which but little light is thrown in medical literature and one that is sooner or later solved by the termination of life. A few persons so afflicted may live through years of a comfortable adult existence—but they live, it seems, regardless of treatment rather than as a result of it.

CHAPTER XIX.

Arteriosclerosis.

DEFINITION AND TERMS.

ARTERIOSCLEROSIS is a progressive degenerative change in an artery which results in a loss of elasticity and in a thickening of the vessel wall, with resultant disordered function of the structures nourished by the affected vessel.

While all three coats of the vessel are involved more or less in the process, the media is that coat which is believed to be the first involved. Those parts of the body with the smallest blood supply show the earliest effect of sclerosis, as is seen in the frequency of early retinal changes.

Atheromatous plaques is a term applied to the sclerosed patches which are found at necropsy in the aorta and in the other large vessels. *Endarteritis obliterans* defines an obliteration, more or less complete, which occurs in smaller arteries and arterioles. The process involves as a usual thing only a short length of vessel, but inasmuch as it narrows the lumen or blocks the vessel completely, it is of clinical importance. The condition is not always pathologic, for it is the process which occurs in the umbilical vessels after they have ceased functioning; it also occurs in the terminal portion of remaining vessels after a part has been amputated. *Endarteritis obliterans* is not an infrequent occurrence during the course of

acute infections. *Capillary fibrosis* is a term applied to such smaller arterial changes as produce the dry and wrinkled skin, the loss of hair, the *arcus senilis*, the disturbances of sensation and of touch, which are seen in persons of advancing years.

CIRCULATORY EFFECTS.

Before considering the effects on the circulation which are brought about by sclerotic changes in the arteries, it is well to pause long enough to recall to mind the varieties of tissue which constitute the three arterial coats. The *adventitia*, or outer coat of an artery, consists of connective tissue in which elastic fibers predominate. The adventitia is thicker in exposed arteries, such as the brachial; it is of comparatively little thickness in arteries which are in a protected situation, as is the abdominal aorta. In the *media* or middle coat there is also elastic tissue, but here transverse muscle fibers occur in greatest abundance. It is in the media that the *vasa vasorum* are distributed—these being the small nutrient vessels in the walls of the larger supply vessels. The smaller vessels are believed to receive their nourishment from the blood circulating within them, as the *vasa vasorum* are not to be found in the media of the smaller vessels. The *intima* or internal coat consists of endothelial cells surrounded by longitudinal elastic fibers and connective tissue. The distribution of elastic tissue and of muscle fibers further varies with the (1) location and with the (2) mechanical function of an artery. For example, the media of the renal arteries contains more muscle fiber than does the carotid, even though the vessels are of approximately the same size.

Arteries are classed as distributing and peripheral vessels. The larger distributing vessels which have the greater mechanical function to perform, are known as "mains," *viz*: the aorta, carotid, brachial, femoral, etc., they are also known as the *semi-elastic* arteries for in them elastic fibers predominate. The peripheral arteries are known as "supply" vessels; in them and in the arterioles, muscle fibers predominate.

The circulatory phenomena arising in sclerosis of either the distributing or the supply vessels are defined by Wiggers¹ as follows: The more nearly the semi-elastic vessels approach the condition of inelastic tubes the more rapidly the pulse is propagated to the periphery. As a consequence of the reduced distensibility, the quantity of ejected blood cannot be accommodated in the aorta and, hence, a larger onward displacement occurs during systole. During the subsequent diastole, however, less elastic or potential energy is available to move the blood onward, hence the flow and pressure both diminish rapidly. The result is a tendency toward an intermittent flow at the periphery and a rapid drop of the pulse wave in diastole.

We may now inquire what effect peripheral sclerosis has on the arterial circulation. Inasmuch as the endarteritis causes a reduction in the lumen of the peripheral arterioles, and resembles an increase in peripheral resistance, the decreased peripheral flow from the arteries to the capillaries causes an accumulation of blood in the arterial circuit and so elevates both systolic and diastolic pressures.

¹Wiggers, Carl J.: "Circulation in Health and Disease," Lea and Febiger, Philadelphia, 1915.

The dynamic effect of sclerosis limited to the large distributing arteries and to the terminal arteries and arterioles is summarized in the following tabulation:¹

SCLEROSIS OF DISTRIBUTING VESSELS.		SCLEROSIS OF PERIPHERAL VESSELS.
Systolic pressure	Increased.	Increased.
Diastolic pressure ...	Unaltered or decreased.	Increased.
Pulse pressure	Increased.	Decreased.
Pulse amplitude	Decreased, unaltered, or increased.	Increased.
Pulse shape	More rapid descent.	More gradual ascent and descent.
Blood flow from arteries to capillaries .	Increased in systole. Decreased in diastole.	Decreased in systole. Decreased in diastole.

ETIOLOGY.

Arteriosclerosis, it may be generally stated, occurs in persons of advancing years. Many of the earlier manifestations of the condition are seen as the meridian of life is approached, often in persons whose history of previous diseases is negative and whose lives have been clean and well ordered. Advancing years so often manifest themselves in arterial change, and arterial change so often accompanies advancing years, that there has arisen as a consequence of this general observation, the trite truism "A man is as old as his arteries." The condition, however, is not at all confined to advancing years, and may frequently be observed in young adults.

Males are more often affected than are females, due perhaps to the more rigorous and more exposed life of men. *Occupation* has a bearing on the develop-

¹ Wiggers: *Loc. cit.*

ment of the condition, for its greater manifestations are in persons of the high-strung type, such as might be represented by the man of large business affairs, by the banker or indeed by physicians, many of whom spend their days in high-tension response to the demands made upon them. The man of brawn, who earns his living with hammer or with shovel, is also a frequent victim of arteriosclerosis. The worker in iron mills, or the lumberman, in whom strenuous physical exertion brings old age prematurely, are further examples of arteriosclerosis resulting from long-continued physical strain. Klotz has stated that medial calcification is more common in the vessels on the right side of right-handed people.

Habits.—It is a frequent clinical observation that abuse of tobacco, which produces *tabagism*, tends to induce arteriosclerosis. This observation must be given weight, despite the fact that there is little experimental evidence to set forth the exact manner in which tobacco acts in producing the condition. *Alcohol*, on the other hand, readily lends itself to logical explanation as a productive factor; if the temporary acceleration of heart rate which follows the ingestion of alcoholic beverages be multiplied several times a day for the period of several years, it can readily be seen that the extra and unnecessary load thus thrown on the circulatory system would result in premature aging of the blood vessels. Another factor of no small moment is *lack of sufficient rest*. The rack and ruin which results from driving a motor continually on high speed, despite the load and despite the road, finds a parallel in the racked and ruined victims of modern high tension living. When the hours

of activity are prolonged far into the night, or when theatre or card parties sustain the interest of a mind that is wearied with the duties of the day, or when dancing sustains the muscular effort in hours which should be devoted to rest, unnecessary wear and tear on the arteries ensues. *Overeating*, with its attendant gastric disturbances and with the additional load thus thrown upon the kidneys in aiding to eliminate the toxins resultant from partially-digested food, is also an etiologic factor. "One of the worst cases of arteriosclerosis I have seen was that of a man aged forty, whose vessels, heart and kidneys were all gravely involved, and whose condition was the direct result of his occupation. He was for years on board a large liner, and it had been his duty to taste all the dishes of the various dinners before they were distributed to the passengers and the crew."¹

Mechanical irritation of a blood-vessel may be a possible cause of arteriosclerosis. The arteries in the arms of a blacksmith or in the legs of a letter-carrier are more apt to be thickened than are the arteries elsewhere in their bodies (Fig. 72). The coronaries, which are frequently invaded by sclerotic changes, may owe their degeneration in some degree to the mechanical irritation to which they are subjected with each contraction of the heart; certainly they are more subject to sclerotic change and more subjected to mechanical irritation than are the vessels in more deeply situated, less mobile organs. *Toxic conditions* such as chronic lead poisoning induce arterial change. The *acute infections* (Fig. 73) of

¹ John Hay, Liverpool: "Some Aspects of the Senile Heart."

rheumatic fever, gout, arthritis and typhoid fever produce at times marked arterial degeneration. Theodore Janeway found plaques of atheroma in the blood-vessels of 21 people out of 52 who died from typhoid fever, and typhoid fever, be it remembered, is more a disease of adolescence than of advanced life. *Focal*



FIG. 72.—MEDIAL CALCIFICATION.
The X-ray reveals calcification of the arteries
of the leg (Cowan.)

infections, such as dental apical abscesses or other foci of suppuration, may be factors in bringing about early arterial symptoms. Among the *chronic infections*, *syphilis* is the one most persistent in its attacks upon the integrity of the cardiovascular system. Several years are believed to elapse between the initial lesion and the advent of circulatory symptoms; but when luetic infection once manifests itself in arterial

degeneration the clinical symptoms seem to progress more definitely and with more rapidity than when vessel damage arises from other causes.

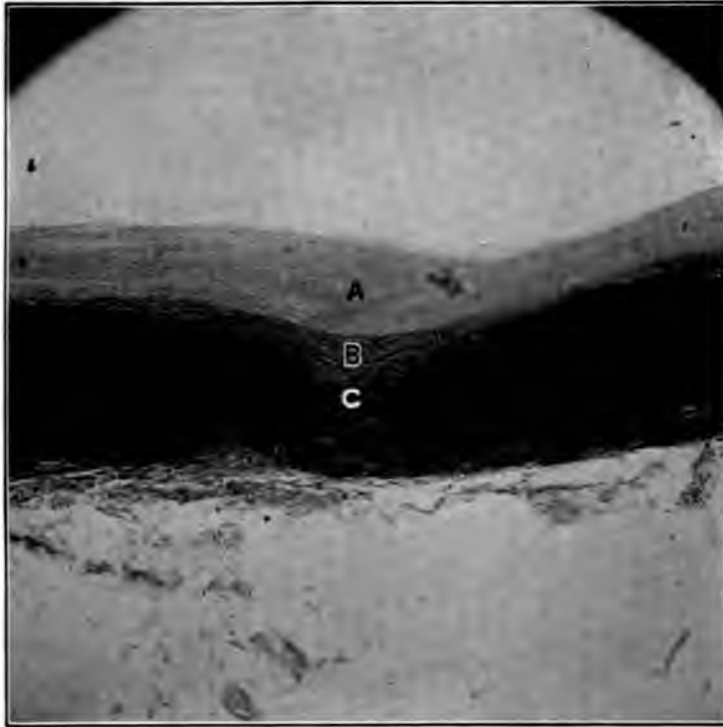


FIG. 73.—BEGINNING ARTERIAL CHANGE.

The photomicrograph shows, in its center, beginning arterial change in a patient who died of influenza in the 1918 epidemic. The thickening of the intima at *A*; the loose elastic tissue at *B*; the condensation of elastic tissue at *C*, all constitute a microscopic arterial plaque. (Courtesy of *Dr. Allen J. Smith*.)

The Ductless Glands.—Sajous,¹ in considering the rôle of the ductless glands in arteriosclerosis, draws attention to the effect produced by toxemia upon the

¹ Sajous, Charles E. de M.: "The Ductless Glands in Cardiovascular Diseases"; *New York Medical Journal*, May 26, 1917.

internal secretion of the thyroid and adrenalin glands. In response to the demands thrown upon them by the toxemia, these glands respond with increased activity and "we witness the phenomena of stimulation—flushed face, brilliant eyes, with perhaps slight precordial pain after an unusually copious meal or unusual exertion, and general vivacity; but it is important to note that this stage of primary exuberance corresponds with the febrile period of an infection, which may, though relatively very short, do as much damage to the blood-vessels as years of overeating, hard labor, etc." Sajous questions the advisability of administering the iodides, or indeed any drug, at this stage, as the addition of the iodides to the thyroiodine which already burdens the organism as a result of thyroid over-activity, may aggravate the malady. "No remedies until the toxic factor, whatever that may be, dietetic, intestinal, bacterial, etc. is eliminated prophylactically." Few indeed are the observing clinicians who cannot subscribe to these truths since Sajous has so originally expressed them; they open a broad field for future clinical research into the etiologic factors of arteriosclerosis.

CLINICAL RECOGNITION.

Arteriosclerosis depends to a great extent for its symptoms on the effect produced in the part or in the organ whose blood-vessels are thickened and whose blood supply is thus diminished, and such symptoms are the symptoms of disordered function of that part. If the vessels of the brain are sclerosed, mental fatigue, drowsiness, loss of memory, confusion and syncopal attacks may precede the rupture of a vessel

in the brain with its consequent symptoms of apoplexy. Should the abdominal viscera be partly robbed



FIG. 74.—ARTERIOSCLEROTIC GANGRENE OF LEG.

Showing the nutritional disturbances that arise from arteriosclerotic occlusion of the vessels. (Jefferson Medical College Museum.)

of their nourishment through arterial degeneration, gastro-intestinal symptoms dominate the picture. When the vessels of an extremity are sclerotic (see Fig. 74), attention is attracted by sensory and thermal changes in the part affected, often with limitations of normal muscular movement and early muscular exhaustion produced by moderate effort. If the kidneys are the organs especially affected, the usual clinical evidences of lack of elimination, absorption of toxins and genito-urinary syndromes come to the fore. Any organ or any combination of organs may be involved; and the more generally the arterial thickening is distributed, the more general are the symptoms.

Frequent Early Symptoms.—A person at the meridian of life may be vaguely conscious that his physique is below par. Instinctively his hours of rest from business become more frequent and his vacations closer together; he also now exercises a care which he was not wont to take in his habits of eating. More attention is paid to exercise and more attention given to elimination than was his custom. He complains, perhaps, of substernal distress following exertion, of slight attacks of indigestion, of an occasional feeling of faintness or of headaches. Memory defects appear at times when he is weary; the speech may halt at intervals and vertigo, transitory numbness and slight ocular disturbances be the symptoms which cause the person with beginning arteriosclerosis to first consult a physician.

The pre-sclerotic stage is a condition of elevated blood-pressure which is believed to exist before the advent of arterial thickening. That it is by no means

always present is quite evident from the clinical experience of many physicians. When it does occur the elevation is often found to be due to toxins or to defective elimination, and generally subsides when appropriate treatment is instituted.

As intimated in a preceding paragraph the onset of arteriosclerotic symptoms is *usually* insidious. Damage to the artery has already taken place before the advent of symptoms, and it reveals itself in consequence of some physical effort, some sustained mental or emotional stress, some strain imposed upon an organ or set of organs—a demand in excess of that to which the physique has been accustomed. For example, overeating might provoke gastro-intestinal symptoms in a person who is bordering upon arteriosclerotic change; the indigestion itself may be of little moment; but occurring at a time in life when thickening arteries are unable to withstand any added strain, symptoms of arteriosclerosis may then manifest themselves and remain established long after the provocative gastro-intestinal symptom has disappeared.

Intermittent claudication may be an early symptom. The term is applied to a sensation of weight or of pain in the leg which is brought about by walking and which produces sudden lameness. It is not provoked by the rapidity with which one walks nor does the distance travelled have any bearing in provoking the symptom. It is believed to arise as a result of the blood-supply to the leg muscles being insufficient for their nourishment, on account of spasm or sclerotic changes in the smaller of the vessels. To the pain, sensation of weight and lameness, there may be added sensory disturbances which the patient describes

with the words "tingling," "numb," "dead." The pain of intermittent claudication is sometimes relieved by placing a rubber band or tourniquet about the upper third of the thigh; by leaving it in place for an hour or two the pain is less likely to recur on further attempted effort.

Blood-pressure.

The systolic pressure in arteriosclerosis is for the most part elevated, although there are types of the condition, such as Albutt's "decreascent" type, to which this observation does not apply. But it is to be remembered that hyperpiesia (high pressure) may be a compensatory process and, that in many instances it may be safely regarded as an expression on the part of nature of an attempt to maintain the circulatory balance necessary for the nourishment of a structure. Elevated systolic pressure is not always an evidence of arteriosclerosis, despite the fact that the two are so often associated; hyperpiesia may arise as a result of temporary causes such as emotion or excitement, and is present in some forms of valvular disease, as well as in many conditions other than arteriosclerosis (see page 131).

Isolated blood-pressure readings are worthless; they are worse than worthless, for they may be misleading. Temporary rises due to emotion, excitement or other causes may occur in the arteriosclerotic subject as well as in a normal person. Therefore, if blood-pressure estimates are to be of significance to the physician, they should be frequently repeated and continued over a period of time. Then they may at times be an index to the value of treatment. The

degree of blood-pressure elevation is no more an estimate of the extent or severity of an arteriosclerotic process than the number of bacilli in sputum is an estimate of the extent of lung involvement in pulmonary tuberculosis.

Kidneys.

One expects to find the kidneys involved to some degree when there is clinical evidence of arteriosclerosis. The frequency of this association has given rise to much academic debate as to which is cause and which is effect—as to whether the kidneys are primarily at fault, or whether the kidneys respond with inflammatory changes to the extra burden imposed by thickening of the renal vessels. It is certain that one sees what one believes to be beginning arteriosclerosis, in which urinalysis affords no evidence of renal damage; and, on the other hand, one sees well-marked laboratory reactions in the urine of elderly people who give little clinical manifestation of arterial thickening. It is possible for kidney disease to induce arteriosclerosis, but it is more probable that arteriosclerosis induces kidney change. This seems the more likely view when one reflects that infections, intoxications, defective elimination and overeating are among the predisposing causes of arteriosclerosis; the kidneys actively concern themselves with elimination of such exciting causes, and consequent damage of kidney structure, with diminished function and damage to the renal vessels would indeed seem a logical course of events.

Uremic manifestations are frequent. They may not occur until some dietetic or other indiscretion has

overtaxed kidneys in which the margin of health has been gradually reduced. It is after such an error in living that an unrecognized kidney affection may reveal itself and uremic symptoms of minor or major degree become a part of the clinical picture.

"*Cardio-vascular renal disease*" is a blanket term which does not describe any definite clinical picture. Cardiac symptoms may predominate, vascular symptoms may predominate or kidney manifestations may rule the situation. It is a co-existence of conditions which is often encountered in advancing cases of arteriosclerosis, and can be brought about by degenerative changes in the smaller arterioles, especially those of the heart and kidneys.

The Heart.

While the heart may be symptom-free in arteriosclerosis, in the majority of cases it will yield some evidence of participation in the sclerotic process of the arteries. Often there are abnormal sensations referable to the precordium, varying from slight precordial uneasiness to the agonizing pains of *angina pectoris*. Frequently there are disturbances of the cardiac mechanism, such as premature contractions, etc., which are described in the following paragraph under "Pulse." When the splanchnic vessels are affected or when there be extensive sclerosis of the thoracic aorta, the *heart enlarges*. Should the heart be increased in size, the cardiac impulse is often sharply defined and more toward the left side than usual; the first sound heard at the apex is prolonged; both first and second sounds at any of the four puncta maxima are usually loud and clear; the second sound

at the aortic area is often accented. There are no murmurs characteristic of arteriosclerosis. As a result of cardiac enlargement apical systolic murmurs may be heard. There is frequently, too, a basal systolic murmur due to intimal roughening of the aorta. Such murmurs lack the associated phenomena which enable one to translate them as valvular disease.

Pulse.

The pulse affords information first, as to the condition of the arterial wall and second, as to any disturbance of rate, rhythm or volume in the cardiac contraction.

Estimation of arterial thickening is usually made from the degree of resistance which the examiner finds in the radial or in the brachial arteries; and it is helpful to acquire the habit of palpating the temporal, carotid, femoral, dorsalis pedis and other available arteries as well. What may seem to be increased resistance in a radial may be a purely local process confined to that artery alone, and thus lead to false conclusions if one is not thorough in the search for information which literally lies at the finger tips. Even though all of the superficial vessels should be found to be infiltrated, their area must indeed be small when compared with the total blood-distributing area in the large splanchnic (visceral) region; with this thought in mind one would not be surprised at the absence of symptoms in such a case, nor would one expect the systolic pressure to be materially raised so long as the splanchnic region remained unaffected.

Various terms are used to define the degrees of infiltration which are found in sclerosed radial or other

superficial arteries, varying from the term *infiltrated*, which is applied to beginning arterial change, up to the term *calcification*, which is used to describe an artery utterly lacking in resiliency on account of a deposit of calcium salts in its substance. A "*locomotor*" artery is one that moves from side to side at each pulsation, as may be seen in a sclerosed brachial when the elbow is slightly flexed and the forearm pronated. The term "goose-neck" artery describes the highly tortuous and thickened vessel of superficial situations. A "*whip-cord*" or "*pipe-stem*" artery is one which rolls under the finger quite in the manner that either one of these materials might do if under a thin investment of skin. "*Beading*" of an artery is the irregular recurrence of hard nodular deposits of calcium salts which greet the finger as it is run along the course of a vessel.

Disturbances of rate in the pulse of arteriosclerotics are manifested in periods of tachycardia which may arise in the absence of any exciting cause. The increased rate is usually transitory and produces but little discomfort. *Disturbances of rhythm* are shown in premature contractions, which may be trivial and cause only a slight disturbance in the rhythm of the pulse during a day, or which may be so freely interspersed in the cardiac contractions as to suggest auricular fibrillation upon casual examination at the wrist. *Disturbances of rhythm and of volume* manifest themselves in auricular fibrillation which may occur in transient periods or which may become permanently established. It is often a terminal event in arteriosclerosis. *Disturbances of conductivity* are revealed in heart-block, usually of the lesser degrees,

although complete heart-block may be an added complication to the senile heart. *Disturbances of contractility* are evidenced in the pulsus alternans; while it may occasionally persist for a period of months, it is usually premonitory of the approaching end of life in arteriosclerosis.

It should be borne in mind that the disorders of the cardiac mechanism just enumerated are neither symptomatic nor diagnostic of arteriosclerosis. When they are encountered for the first time in this condition, they are of significance in that they indicate that the heart structure is manifesting the burden of extra labor or of toxins that are being imposed upon it; or they may indicate that heart tissue has been damaged by the same degenerative process which sclerosed the arteries.

The Eye.

Arcus senilis does not now have the same significance which was at one time attached to it as a sign of arteriosclerosis. This greyish ring, which forms at the periphery of the cornea, is the result of a colloid degeneration of some of the corneal layers. It is observed at times in persons who are apparently normal, as well as in elderly persons with arterial change. The *retina* does not always show changes in its arteries, but it shows them sufficiently often and with sufficient constancy to be considered of value as an early diagnostic sign in arteriosclerosis; in some instances these changes are the fore-runners of other clinical manifestations. Physicians who are not within easy call of an eye consultant would do well to familiarize themselves with the simple technique of

the ophthalmoscope, and to employ it in the vague complaints which so often herald the approach of sclerotic changes in the arteries. The usual findings are that the vessels of the retina are irregular in caliber and the veins are contracted at those points where they are crossed by infiltrated arteries. The retinal arteries may be more tortuous than usual and the central light streak is often very bright, giving the arteries a luminous appearance to which the apt term "silver wire arteries" has been applied. Obstruction of the central artery or vein may also be observed and progressive optic atrophy may be revealed by the employment of the ophthalmoscope.

To tersely summarize the preceding pages on the clinical recognition of arteriosclerosis, one may conclude as follows. In a patient past the meridian of life, who complains of fatigue on moderate exertion, dizziness, confusion, precordial distress, palpitation, cold extremities, insomnia, bronchitis, anginal symptoms, slight edema, and in whom the systolic pressure be found to be constantly from 30 to 60 mm. higher than the average pressure for a given age, arteriosclerosis is the probable diagnosis; but further inquiry should be conducted along the lines outlined on the preceding pages, to arrive at a definite opinion.

The *x-ray* finds some diagnostic employment in the condition when requisitioned to establish the cause of such symptoms as would arise from medial calcification of certain arteries (Fig. 72).

PROGNOSIS.

Arteriosclerosis is not a condition in which pathologic tissue change can be restored to normal; nor is

it a condition the progress of which can be arrested, except in unusual instances, for it is essentially progressive in nature. The physician may, by the institution of a proper therapeutic and dietetic regime, so relieve the burden imposed upon the circulation and upon the organs of elimination that the patient is kept comfortably unconscious of the progress of his condition. Indeed, such measures have been credited with adding years to the expected tenure of life.

The prognosis in arteriosclerosis is to a considerable extent based upon the etiologic factors in the condition. If the symptoms be mild in character and are those attributable only to the advance of years, the outlook as to relief of symptoms and additional years is of course more hopeful than if the arterial degeneration were the result of toxic influences, or due to degenerative changes induced by long continued infections. The prognosis is also dependent upon the response to treatment which the patient exhibits under rest and elimination. If under proper management annoying symptoms disappear and the general tone of the patient improves, the prognosis as to years is again more favorable than if treatment were unattended with results.

When the condition is the result of absorption from some long-unrecognized focus of suppuration, the institution of operative procedures for the evacuation of pus or destruction of pyogenic membrane may arrest what would otherwise be progressive arterial change. If it be found to be due to a definite systemic infection such as syphilis, appropriate treatment may succeed in checking the progress of sclerosis.

Should advanced arteriosclerosis be announced by the occurrence of cardiac dyspnea, apoplexy, pulsus alternans, Cheyne-Stokes' respiration and other serious symptoms, the prognosis is ominous.

TREATMENT.

The first step in the treatment of arteriosclerosis, as in the treatment of any condition, is to *seek for and if possible remove the cause*. Underlying infections (see *Etiology*), either constitutional or focal, are to be appropriately treated with the indicated remedies or by operative procedures. For example, a luetic history or more than one positive Wassermann reaction, point the way to specific treatment; or pyuria may be an indication of a long-standing kidney affection which the *x*-ray may suggest is best relieved by nephrolithotomy. Other focal infections have already been mentioned on page 224. The constitutional conditions enumerated on page 36 should likewise be sought for. In such a thorough manner should the physician continue the search for a cause, possibly remediable, which is aggravating the arterial condition.

The early symptoms of arteriosclerosis, if they be considered one by one, are of little moment; taken in the aggregate however, they so reduce the physical and perhaps the mental efficiency of the patient that it is necessary to change the manner of living or risk a shortening of life. There is a border-line between the life of activity and the life of retirement which all must sooner or later reach. Such minor symptoms as an enforced curtailment of accustomed exercise, shortness of breath on slight exertion, dizziness, con-

fusion, mild digestive disturbances, ocular defects, irascibility, or lack of interest in the activities of life which have heretofore proved engrossing, are frequent manifestations of early arteriosclerosis and they mark the border-line between the life of activity and the life of partial retirement from business. It has well been said that the person so afflicted is confronted with the same problem as that which presents itself to the teamster who is taking a load up a hill, the grade of which is ever increasing: the problem is whether to apply the lash or lighten the load. While it may be necessary at times to employ both expedients, manifestly the first choice is to lighten the load when the end of the road is not yet in sight. In treating a patient with arteriosclerosis the load is lightened by *relaxation*, by *rest* and by *elimination*.

Relaxation.—The gospel of relaxation is perhaps the most difficult subject which the physician is called upon to teach. It is no easy task to persuade an active man whose strong, aggressive personality has long dominated his field of business that his symptoms, trivial as they may seem to him, necessitate a change in his method of living—a relaxation from activities and cares, and the devoting of many hours to physical and mental rest. Such men rarely acknowledge that they have physical limitations, and never admit to the confusion of thought or slowing of mental processes which may be quite apparent to business associates. Hence it is only by persistence, by insistence and by constant repetition that the physician can hope to impress the active man of affairs with the degree of care necessary for his well-being and for the lengthening of life.

Short vacations or occasional holidays may bring marked benefits which, however, soon are lost when the patient returns to his accustomed manner of living. Hence, the hours spent in business activities should be shortened; more time should be spent at the table; more hours should be given to moderate exercise in the open air; sufficient time for personal hygiene should be taken in the mornings, supplanting the hurried rush to the office; more hours should be devoted to sleep. Physical and mental relaxation are the best preventives of possible rupture of a vessel at the base of the brain, with its attendant stroke of apoplexy.

Rest.—There are instances in which the physician will desire to put the patient at complete rest in bed in order that he may more fully study the symptoms; he may also thus anticipate possible emergencies which might arise should the patient continue active and busy with his affairs. Further, with the patient absolutely under control in bed, the physician will be better able to estimate the beneficial results of treatment and thus arrive at a more definite prognosis.

It is the part of discretion, when the severity of the symptoms warrant, to put the patient to bed at the *beginning* of a course of treatment and, as his improvement progresses, allow him to be up and around the house, rather than to later attempt to impose bed restrictions on a patient who has once been permitted to be ambulant. The patient who has been confined to bed for a period varying from a few days to a few weeks will certainly be spared the strain that physical activity would throw upon his damaged circulatory system. His diet will receive more regu-

lating than if he were tempted by the foods served at the family table. Furthermore, elimination, which is so essential to proper treatment, will receive the attention which its importance warrants if a nurse or skilled attendant carries out the physician's instructions for the patient who is being treated in bed.

Elimination.—When one recalls that the kidneys are frequently involved to a greater or less degree in arteriosclerosis and that the retention of toxins due to this cause may aggravate the symptoms the significance of elimination becomes at once apparent. The curious theory has been advanced in some quarters that increased fluid intake in arteriosclerosis adds to the volume of fluid within the body and thus increases the work of a damaged circulatory system. Whatever theoretical foundation there may be for such an idea, the fact remains that patients are quite invariably benefited by an increase in the fluid intake. When drinking water is used for the purpose of flushing it should of course be thoughtfully administered, and taken in quantities of one or two glassfuls, at a time when the stomach is empty, *viz*: on arising, half an hour before meals, three hours after, and on retiring. There may be instances in which the infusion of digitalis is indicated as a diuretic; it should be prepared with the precautions suggested on page 209. Such an instance exists when it is desired to remove dropsical effusions which have accumulated in the body by increasing the urinary output. Under such circumstances the Karrel diet (page 211) should also be employed and the water intake for the time limited. Sweet spirit of niter and potassium citrate may be

employed as a diuretic in the proportions of the following prescription:

R Spiritus ætheris nitrosi f℥ss.
 Potassii citratis ʒij.
 Aquæ destillatæ q. s. ad f℥iv.
 M. Sig.: Two teaspoonfuls in ½ glass of water at 4-hour intervals.

The *bowels* are best regulated, not by the use of hydragogues which by their excessive action may induce exhaustion, but by salines in moderate daily dose. The milder laxatives such as senna, compound liquorice powder, cascara sagrada, etc., may be preferred when it is desirable to stimulate peristalsis over a considerable length of time.

The *skin* is kept active by the use of the daily tepid bath which may be followed by witch hazel rubs. Massage may also be employed in order to stimulate elimination by the skin. Hot baths or Turkish baths, if used at all, are to be cautiously employed and only under the direct supervision of the physician.

As has already been stated, there are cases of arteriosclerosis in which milder uremic symptoms are manifest. For the emergency of uremic convulsions or uremic coma which may arise the usual treatment, consisting of purgation, hot packs, free elimination and support for the heart if failing, are of course indicated.

Dict.—Food should be regulated, both as to its quantity and as to its constituents. As a general proposition, the protein intake should be limited and meats, condiments, sugars, fats and stimulants interdicted. No hard and fast dietary rule can be formulated that is adaptable to every case. It is well to re-

member in this connection that there are persons who have learned from years of experience to avoid certain foodstuffs to a degree that would seem to deprive them of necessary elements, and to use other foodstuffs to a degree which might seem excessive. Yet they have thrived on their continued dietetic vagaries. Therefore, when the physician arbitrarily insists on a patient eating certain foods to which the patient has a natural repugnance or which he confidently believes will do him harm, the physician by such insistence commits an error in judgment in many instances. It is also no less an error in judgment to insist that the patient abandon certain foodstuffs which, while questionable, are not *definitely* harmful and upon which he has subsisted for years. For the first few days in bed liquid or semi-liquid diet may be advantageously employed in order to avoid overtaxing the system and to encourage elimination; afterwards a more liberal diet may be permitted. Foods which contain a *high* percentage of protein should be avoided. (as far as it is feasible to avoid them), familiar examples of which are red meats, eggs, fish, cheese, peas and beans. Perhaps the physician will desire to employ, for a time, the diet for the "Senile Heart," as given on page 386.

DRUGS.

It should be remembered that *relaxation, rest, elimination* and *diet regulation* are the four cardinal principles of treatment which reduce high blood-pressure in the great majority of instances, and they reduce it naturally and safely. In arteriosclerosis elevated systolic pressure (hyperpiesia) is often com-

pensatory; indeed it is probably nature's only method of getting sufficient nourishment to some organ or tissue of the body which might otherwise be more or less ischemic, owing to capillary fibrosis in the part. If this view be tenable, then the administration of "circulatory sedatives," such as veratrum, might well be referred to as meddling therapeutics. Vasodilator drugs, on the other hand, may be demanded in an emergency, as when apoplexy threatens, but it is not wise to employ them as a rule unless elimination is being coincidentally practised. Drugs of the nitrite group are the vasodilators which are employed in the reduction of high pressure by drugs. Urgent symptoms may demand amyl nitrite in 2 minim pearls, crushed in the handkerchief and inhaled. *The spiritus glycerylis nitratis* is a 1 per cent. aqueous solution of nitroglycerine and is given in 1 or 2 minim doses, sometimes gradually ascended. Tablets of nitroglycerine each contain 0.01 of a grain. In administering the nitrites it is well to remember that their action is fleeting in character, the reaction not being sustained, and that for this reason they should be administered at three- or four-hour intervals.

Digitalis.—This drug has no place in the *routine* treatment of arteriosclerosis. Its loose administration as a universal panacea for any affliction of the heart or circulation cannot be too strongly protested against. In arteriosclerosis there are two *exceptional* conditions under which it may be indicated; the first indication is as a support for the heart when cardiac failure threatens as a result of myocardial exhaustion. The second is in those instances where the physician is unable to relieve peripheral resistance

and feels that the resultant strain upon the heart muscle may result in failure of that organ; digitalis is then used to fortify against such a contingency.

Potassium iodide has for years been the sheet-anchor in the drug-treatment of arteriosclerosis. The action of the drug is in doubt, but its effects are not in doubt. It produces beneficial results which are not at all to be explained on the assumption that the drug benefits only syphilitics. One would do well indeed to bear in mind, especially in these days when the pendulum of etiology has swung too far to the side of syphilitic infection, that not *every patient who is benefited by potassium iodide is a syphilitic*.

Balfour thought that the iodides dilated the arterioles; Burnett claimed that they increased elimination; either physiologic action would lower arterial pressure. Modern laboratory experiments have so far neither affirmed nor denied either of these opinions. The administration of the drug today is empirical, just as it has been for generations past. The usual dose of iodide of potassium for long-continued administration is 5 to 10 grains, held in solution in a pleasant vehicle such as compound syrup of sarsaparilla to disguise its objectionable taste, or well diluted in water, taken 3 or 4 times daily, preferably after meals. The administration over a long period of time may bring on the symptoms of *iodism*, the early manifestations of which are coryza, a metallic taste in the mouth, moderate increase in salivary secretion, tenderness of the gums or of the teeth and slight nausea. The appearance of such symptoms is an indication for the withdrawal of the drug. In this connection the warning of Sajous on the subject

of "The Ductless Glands" (page 286), should also be borne in mind.

Electricity in the form of high frequency currents often lowers peripheral resistance and thus lowers the pressure in early and selected cases of arteriosclerosis. Its employment is better left in the hands of the electrotherapist.

Venesection may be of value in warding off such a crisis as apoplexy or heart failure in arteriosclerosis. It is contraindicated in anemic patients and also where there is marked renal involvement.

CHAPTER XX.

Aneurism.

DEFINITION AND VARIETIES.

AN aneurism is a circumscribed dilatation of a blood conducting structure. While aneurisms more often occur in the large arterial trunks, such as the aorta, subclavian, carotid, etc., they may arise along the course of any of the 468 arteries in the body; they may involve the capillaries and, as in the case of a *nexus vasculosis*, involve both capillaries and veins. Aneurisms of the heart have been found at necropsy, as have also aneurisms of valve-leaflets.

Aneurisms are subject to many classifications. They are said to be *primary* at the time of their first appearance; should they recur, as in the case of a femoral aneurism which has disappeared under compression treatment, they are termed *secondary*. A *true* aneurism is one in which the sac is formed by the arterial walls, at least one coat of which remains unbroken; a *false* aneurism is one in which the wall is formed by surrounding structure, the coats of the artery having given completely away.

Further classification is made according to shape. A *sacculated* aneurism is one whose opening into an artery is smaller than the diameter of the sac—a very frequent clinical type. The *fusiform* variety is of spindle shape. A *dissecting* aneurism is one which burrows between the coats of the vessel.

Aneurisms may be simple or *combined*; a combined aneurism is one in which some coats are ruptured and other coats are dilated. A *diffuse* aneurism is one in which all the arterial coats are ruptured.

A *surgical* aneurism is one in which there is a possibility of surgical intervention for its relief. The term *traumatic* is applied to those tumors of blood vessels which arise as the result of trauma or exertion. *Embolic* aneurism forms as the result of dilatation occasioned by the lodging of an embolus.

Further subdivisions are made according to the names of the observers who first described them. No less than seven men have their names attached to tumors of blood-vessels which are known as Potts' aneurism, Rodrigues' aneurism, etc.

Classifications can be multiplied almost indefinitely. This discussion will be confined to the most common clinical variety of aneurism, which is most frequently accompanied by disturbances of the heart and circulation, *viz*: *aneurism of the thoracic aorta*.

ETIOLOGY.

The patient and laborious researches of Warthin¹ were of much etiologic significance in establishing syphilis as a probable cause of the majority of aneurisms of the thoracic aorta, yet syphilis is by no means the solitary cause of aneurism. It should be remembered that there are many instances in which aneurism has arisen as a result of acute rheumatic fever, alcoholism, injuries and as the remote result of acute infections. The presence of aneurism is no warrant for fastening the stigma of syphilis upon the sufferer.

¹ Warthin: *Loc. cit.*

Aneurisms are more frequently encountered during early middle life. They are six times more common in men than in women. Occupation does not predispose to their occurrence, although violent exertion or long continued strain may rupture a vessel coat and precipitate urgent symptoms in a hitherto unsuspected case.

In considering the etiology of aortic aneurism, it is well to trace the condition from the period of initial inflammation to that point where the infection has so weakened the arterial coats that their dilatation or rupture results from the force exerted by the blood-stream on the weakened vessel wall. While aneurisms may develop suddenly as the result of muscular effort, severe paroxysms of coughing, physical strain, etc., careful questioning of such patients will often elicit a previous history which is suggestive of aortitis or of aneurismal dilatation. If the physician is to be of the greatest service to his patient it is necessary for him to recognize that there is such a condition as a "pre-aneurismal stage" of aneurism and that this formative stage may give symptoms of acute aortitis or symptoms of dilatation of the arch, by the early recognition of which one may hope to prevent eventual rupture of the blood-vessel walls with well-nigh hopeless aneurism formation.

ACUTE AORTITIS; THE PRE-ANEURISMAL STAGE.

Those acute infections which so often induce myocarditis and endocarditis are factors in the production of acute aortitis, namely, scarlet fever, typhoid fever, pneumonia, acute rheumatic fever, severe or long-

continued tonsillar infections, septicemia, and even influenza (Fig. 73). Necropsies on patients who have died of these conditions show areas of degenerative change in the aorta. They may not be recognized by the unaided eye, but they can be recognized by the use of various stains which bring out the tissue degeneration under the microscope. Thus it is proven that the aorta may share in the damage which is wrought in heart tissues by these infections, and as a consequence an initial aortic damage arises which becomes the vulnerable point in the integrity of the aortic wall in future years. In this connection one might again refer to the statistics of Thayer¹ who found plaques of atheroma in the aorta of 40 per cent. of 52 cases of typhoid fever.

In considering the etiology of *subacute* forms of aortitis one should not lose sight of the frequency with which the treponema of syphilis invades the aortic arch—a frequency so marked that the aorta should be carefully studied for symptoms and signs in any patient who presents evidence of syphilis elsewhere in the body. As a result of the close association between acute aortitis and syphilitic manifestations, there has arisen the term *aortitis syphilitica*.

SYMPTOMS OF AORTITIS.

(1) *Pain* under the sternum is the most suggestive symptom; with it may be associated a sense of substernal oppression. (2) *Dyspnea*, which need not especially follow physical effort, is often complained of. (3) *Vertigo* may be present in a moderate degree. (4) One or both arms may present symptoms of sen-

¹ Thayer: *Loc. cit.*

sory disturbance or of pain. (5) *Fever* due solely to aortitis, if present at all, amounts to only a slight rise. (6) *Cough* may be added to the clinical picture.

PHYSICAL SIGNS OF AORTITIS.

The force exerted by the column of blood which is ejected from the heart at each systole of that organ increases the strain which inflammation has thrown upon the aortic wall; as a consequence, *dilatation of the aorta* is likely to arise. Carotid pulsation is one of the manifestations of aortic dilatation under such circumstances, but a more valuable sign is the one described by Potain which consists in upward displacement of the subclavian arteries so that they are seen to pulsate directly above the clavicles. These vessels are raised from their normal situation into the position where their pulsations can be seen as a result of the aortic dilatation, which lifts them from their usual position. If one hand be placed at the upper part of the sternum and the other hand be laid on the back between the scapulae, pulsations may be noticed which would otherwise have escaped detection on inspection. Again, if the ear be laid upon the chest wall at either of these points the systolic pulsation may be detected by thus combining the sense of touch and hearing. There may be an increase in the area of aortic dullness noted upon percussion. The total transverse arch of the aorta in health usually measures 5.5 centimeters in the second interspace to the right and left of the sternum, but if aortic dilatation be present that distance is often found to be increased on percussion.

The aortic second sound is likely to be ringing in character. An aortic systolic murmur may be present should the aortic valve be involved; or the systolic murmur may arise as the result of roughening of the internal arterial coat. A systolic murmur is of no diagnostic value in acute aortitis, aortic dilatation or aortic aneurism, except in so far as it co-exists with and is confirmatory of other signs. A diastolic impact may be felt in the second interspace to the right or left of the sternum.

DIAGNOSIS OF AORTITIS.

The occurrence of the above mentioned *symptoms* during the course of or subsequent to an acute infection is cause sufficient to make the provisional diagnosis of acute aortitis. There is no way of definitely determining the condition by *physical signs* unless aortic dilatation be present. In any event, the *x-ray* should be employed to confirm the suspicion of aortitis or aortic dilatation, and may reveal an increase in the diameters of some portion of the aortic arch where physical signs gave no information. Clinical evidence of syphilis or a positive Wassermann reaction in a given case is always cause for suspecting either aortitis or aortic dilatation or both. *Treatment* is undertaken for the purpose of minimizing the inflammation by *rest in bed*, and the employment of such measures as may be appropriately selected from those detailed in the consideration of myocarditis, page 206.

THORACIC ANEURISM.

For the convenience of clinical description of aneurisms which might arise therein, the thoracic

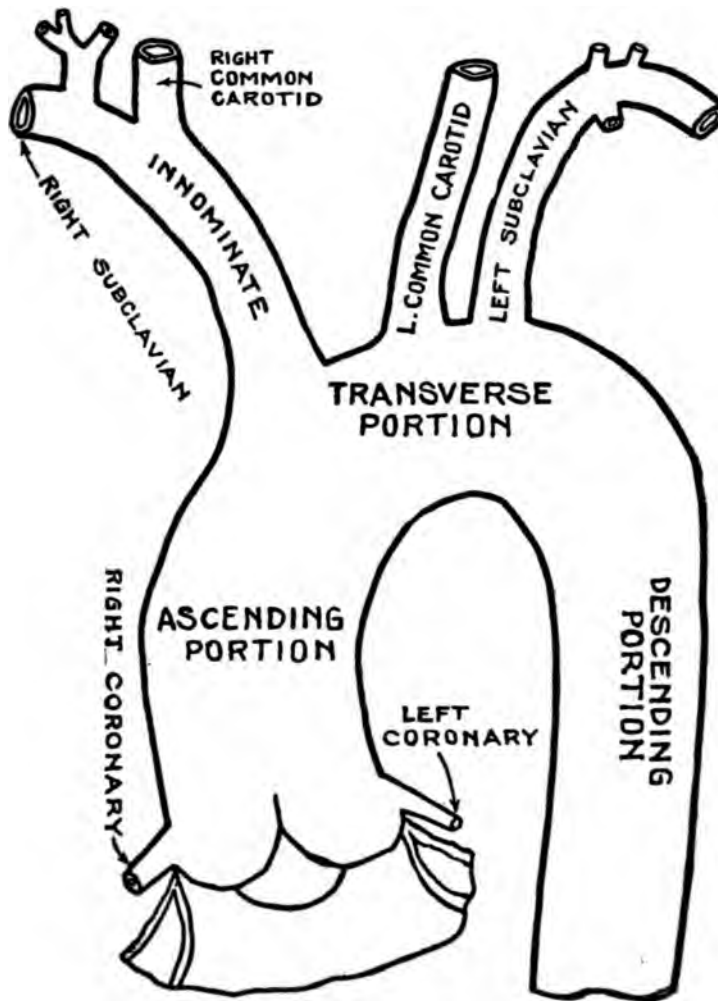


FIG. 75.—THE ARCH OF THE AORTA.
(Natural size.)

Showing areas at which aneurisms may arise and occasion
special pressure symptoms on arterial trunks.

aorta is divided into four parts, *viz*: (1) *the ascending arch*, which is that portion extending from the left ventricle to the innominate artery (see Fig. 75); (2) *the transverse arch*, which extends from the innominate to the left subclavian artery; (3) *the descending arch*, which extends from the left subclavian artery to the level of the fourth thoracic vertebra; (4) *the descending thoracic aorta*, which is that portion of the vessel between the fourth vertebra and the diaphragm.

Aortic aneurism occurs in order of frequency with the clinical divisions just mentioned. Aneurisms of the ascending arch are far more frequent than are those of the transverse arch, while less in respective frequency are those of the descending arch and the descending thoracic aorta.

GENERAL SYMPTOMS OF THORACIC ANEURISM.

Aneurisms have many symptoms in common, irrespective of that portion of the thoracic aorta which may be involved. There are other symptoms especially referable to each of the four clinical divisions of the vessel. First to be considered are those symptoms and signs which are applicable to thoracic aneurism in *general*.

(1) *Pain*.—The pain is sharp and acute if a nerve be pressed upon and is boring in character if a bone be implicated. Paroxysmal attacks simulating angina pectoris may occur.

(2) *Dyspnea* on slight exertion.

(3) *Abnormal Pulsations*.—These may be found in the second interspace, in the suprasternal notch, along the right border of the sternum, or in the back

between the scapulae. Such pulsations are not to be confused with the abnormal precordial pulsations or with the arterial throbbing which are often observed in emotional or neurasthenic persons. *Protrusion* may be noted in any one of the situations just mentioned. Aneurismal swellings exhibit, as a differential characteristic from tumors due to other causes which might be in the same location, the phenomenon of pulsation, usually laterally expansile. Pulsations not noticed on inspection may be detected upon palpation.

(4) *Systolic Thrill*.—The systolic thrill of an aneurism is occasioned by the swirling of blood which takes place when the fluid enters the dilated portion of the vessel from an opening of smaller caliber.

(5) *Diastolic Shock*.—The diastolic shock often present in aneurism is probably caused by the elastic recoil of the aneurism walls following systolic dilatation. A loss of elasticity of the vessel wall or the formation of a laminated blood clot, often miscalled "a mattress of fibrin,"¹ in a portion of a sac may account for the absence of diastolic shock in some instances.

(6) *Other general symptoms* which may be present include: inequality of pupils, due to irritation of the sympathetic nerves; engorged veins of chest, due to intramediastinal pressure; dysphagia, the result of pressure on the esophagus; occlusion of a bronchus, causing an atelectatic lung; noisy breathing, in consequence of a partial paralysis of the recurrent

¹ Following recent studies, Dr. Allen J. Smith is of the opinion that the formation which may occur along the wall of an aneurism can in no wise be considered fibrous tissue; it is a laminated blood clot, containing lime, fat, red cells, fibrin, and a substance resembling amyloid material.

laryngeal nerve; tubular breathing, the result of the conduction of sound from a bronchus by an aneurism.

Enlargement of the heart, long considered a classic sign of aneurism, is found in less than 1 per cent. of cases, according to Howard's autopsy statistics.¹ The heart may of course be pressed downward and to the left to accommodate the new growth within the chest.

EFFECT OF THORACIC ANEURISMS ON PERIPHERAL CIRCULATION.

If the heart has not been affected by the same condition which diseased the artery, the peripheral circulation is not affected by the interposition of an aneurism in the course of an artery. The output of the heart is unaltered; hence the peripheral flow is unchanged, as was shown by the experiments of Stewart, who observed that when the pulse is smaller on the left side, the blood flow is nevertheless almost equal in the hands.

PRESSURE SYMPTOMS RELATIVE TO SITE.

An aneurism makes pressure on certain structures during its growth, and such pressure symptoms vary with the site of the tumor, thus often enabling one to locate the site of an aneurism by symptoms specially referable to some portion of the aorta (see Fig. 75).

(1) *Ascending Arch*.—This is the aneurism which Broadbent designated "the aneurism of physical signs." It produces more displacement of the heart to the left than would a swelling of similar size situated on any other portion of the aorta. On account

¹ Howard: Johns Hopkins Bulletin, III, 266.



FIG. 76.—ANEURISM OF THE AORTA.

The usual diameter of the aorta is seen in the descending portion on the right. It opens into a large aneurism, which in turn communicates with the heart. (Jefferson Medical College Museum.)

of its proximity to the valve it is frequently accompanied by signs of aortic valve leakage. By the pressure which it makes upon the recurrent laryngeal nerve it may produce alterations in the voice varying from a brassy cough and hoarseness to complete aphonia.

If an aneurism of the ascending arch be on that portion of the vessel which is within the pericardial sac (Fig. 76), attacks of dyspnea and anginoid pains are marked, the reason for this being that the pericardial space is lessened by the interposition of the aneurism and the heart action thus interfered with. If the tumor be large within the pericardium, it impedes the entrance of venous blood into the heart and as a consequence venous engorgement is present in the neck and arms. If the aneurism is situated on that portion of the ascending arch which is external to the pericardial sac, the superior vena cava may be compressed with resultant engorgement of the neck, face and arm on the right side.

(2) *Transverse Portion of the Arch.*—This is the aneurism which Broadbent called “the aneurism of symptoms,” for with an aneurism so situated the symptoms are often more marked than are the physical signs. This tumor frequently points backward. It presses the trachea and the esophagus, thereby giving rise to respiratory symptoms, to alterations in the voice and to difficulty in swallowing—which will vary in degree in accordance with the size of the tumor.

Dyspnea and dysphagia may be due to direct pressure on the structures concerned; or they may result from pressure upon the left recurrent laryn-

geal nerve, which is subject to pressure by an aneurism in this situation.

The pulse exhibits more alterations in an aneurism of the transverse arch than in one in any other situation along the aorta. If it presses upon or involves the innominate artery (see Fig. 75), the left common carotid or the left subclavian artery, changes appear in the carotid or radial pulse. If the innominate artery is involved the right carotid pulse is often lowered in volume or delayed, as is also that of the right radial artery. If the left subclavian or adjacent common carotid artery is pressed upon or involved, the left carotid pulse is lowered in volume or delayed, as may also be the pulse in the left radial artery. In estimating delay or retardation of the radial pulse it is wrong to time it by the precordial impulse, for the ventricular contraction naturally occurs 0.1 of a second earlier than the radial pulse. The delay in radial pulsations is estimated by grasping both the right and left wrists at the same time. Manifestly delayed pulsation is a sign that is not constantly present, for it depends upon the location and size of the tumor of the transverse arch; if the aneurism be small and situated on the *concave* portion of the arch it would not be expected to press upon the vessels which arise from the convex surface of the arch. Even though a radial pulse is found retarded, one should satisfy oneself that other symptoms of aneurism are present and not depend on this sign alone, for reasons enumerated in the following paragraph.

Local Factors in Radial Pulse Obliteration.—The pulse is not infrequently absent in the right wrist of well muscled men who earn their living with their



FIG. 77.—ANEURISM.
(Courtesy of *Dr. Elmer H. Funk.*)



FIG. 78.—X-RAY PHOTOGRAPH OF FIGURE 77.

"Large aneurism of the first portion of the ascending arch. It points directly forward. Another aneurismal sac on the descending aorta, more posteriorly. There are large atheromatous plaques in the descending arch." (Courtesy of Dr. Willis F. Manges.)

arms. Persons in whom broken bones of the wrist have undergone repair, may possess a radial artery that is not palpable. Old inflammatory processes at the wrist joint may obscure the radial pulse. Obliterative change further up in the course of the artery may prevent a pulsation from reaching the wrist. There are many persons in whom the radial artery departs from its superficial location and lies so deep as not to be perceptible at its usual situation along the styloid process.

(3) *Aneurism of the Descending Aortic Arch.*—

An aneurism in this situation, between the subclavian and fourth thoracic vertebra, is most frequently directed along the spine and may point in the back. Thus it may press on nerves of the spinal cord at their point of exit and cause pain in the part supplied by these nerves, such as the shoulders or the axillary lines. Enfeebled or delayed femoral pulsations may be noted and perhaps there will be an absence of pulsation in the abdominal aorta.

(4) *Aneurism of the Descending Thoracic Arch.*

—The contact of such an aneurism with the spine may result in erosion of the lower thoracic vertebra. As a result pain of an excruciating character may be elicited if the vertebral column be pressed upon.

PHYSICAL SIGNS OF THORACIC ANEURISM.

Inspection.—Abnormal systolic pulsation may be noted between the clavicle and third rib; it may be suprasternal or it may be found in the interscapular region in the back. Feeble pulsations may be noticed, these occurring oftener to the right of the sternum. The pulsation may be lost altogether if the patient be

examined in the erect posture alone. It is a good rule to routinely examine the anterior chest wall with the patient in the recumbent posture, a light falling directly upon the chest and with the eyes of the examiner on the level of the chest wall.

Palpation.—If the thoracic wall has been disintegrated a definite pulsating tumor, brawny and indurated, will protrude (Fig. 77), and the maximum cardiac impulse may be displaced to the left by pressure of the new growth. Aneurismal “erosion” should not be attributed to the mechanical effect of a pulsating tumor upon bone substance. It is due to an extension of inflammation from the walls of the sac, it having been shown by Smith¹ that in the case of an aneurism due to syphilis, the *Treponema pallidum* was the destructive agent in sternal “erosion.”

Tracheal tugging may be elicited, especially in aneurism of the transverse arch. This sign, first described by Oliver, is not pathognomonic of aneurism, for it also occurs in aortic dilatation. It is elicited by having the patient, who is in the erect position, elevate his chin; the cricoid cartilage is then grasped between the finger and thumb and gently pressed upward. A tug will be felt at each pulsation of the heart, due to the fact that the arch of the aorta is crossed by the bronchus and, if the aorta be dilated, the bronchus moves with each systole of the heart.

Resonance is diminished over the area occupied by the tumor. This may be noticed especially in reference to the ascending aorta, which is very accessible to percussion. Diminished resonance will change

¹ Smith, Allen J.: New York Med. Jour., March 7, 1914.

into dullness and flatness as the aneurism increases in size.

Auscultation reveals nothing distinctive in aneurism. A diastolic sound may be present on either side of the sternum and is co-existent with the diastolic shock previously mentioned. The systolic murmurs which are quite usually heard in aneurism are of no diagnostic significance.

DIAGNOSIS OF THORACIC ANEURISM.

When an aneurism has advanced to the stage where it presents as an external tumor, or even to the stage where it causes distinct pulsations in the interspaces, in the suprasternal notch or at other superficial locations, the diagnosis is evident. Should confusion arise with other tumors in similar locations, the distinction can be made by remembering that an aneurism is a pulsating tumor, the pulsations of which are synchronous with the systole of the heart, and the pulsations are laterally expansile in character. There is, however, little benefit to the patient in diagnosing an aneurism after it has progressed to this stage (Figs. 77, 78, 79, 80). The diagnosis should be made long before such an unfortunate event occurs.

Reliance should not be placed upon the presence of physical signs, nor should the absence of any of the signs enumerated in this chapter disarm a suspicion of present or future aneurism, which suspicion has arisen as the result of constant substernal pain, substernal oppression, dyspnea, cough and palpitation. There is only one way to make a definite diagnosis of early aneurismal formation. The *x-ray*

should be employed as the only dependable means of early diagnosis.

In employing the Röntgen light, *fluoroscopic* examination is that form which is of special advantage in the diagnosing of aneurism. It permits of a searching examination of the mediastinum from all angles and directions. If an aneurism be detected, the fluoroscope permits one to estimate the degree of pulsation in the sac; it shows the area of greatest pulsation, thereby allowing one to anticipate the source of future pressure symptoms should the aneurism progress. Later on, it furnishes some basis for an opinion as to the elasticity of the vessel at that point where diminished expansion might suggest the possibility of early rupture of the elastic arterial coat.

Early fluoroscopic recognition of an aneurism offers the best hope to the patient so afflicted. It means the early institution of treatment, such as the curtailment of activities, enforced periods of rest and treatment of the underlying cause, and thus holds out hope of preventing irreparable structural damage which may ensue if one depends for the recognition of aneurism upon the presence of clinical symptoms alone.

PROGNOSIS OF THORACIC ANEURISM.

Some beginning aneurisms which are recognized by fluoroscopy never reach a greater stage of development than that in which they are first seen, owing to the institution of proper treatment and change in the habits of life. Aneurisms which seem to progress in the absence of treatment have been observed by Vaquez and Vordet to actually reduce in size follow-



FIG. 79.—ANEURISMAL "EROSION."

The upper part of the sternum has completely disappeared, as a result of extension of inflammation from the aneurism underneath. (Jefferson Medical College Museum.)



FIG. 80.—ANEURISMAL "EROSION."

Side view of Figure 79. A section has been cut from the aneurism, showing the thickness of its wall. (Jefferson Medical College Museum.)

ing several arsphenamin injections which were directed to the cure of the causative infection, syphilis. These same observers report that the fluoroscope has shown, under such circumstances, an actual reduction in calcified areas in sacculated aneurisms of the ascending and transverse portions of the aortic arch.

As to the prognosis of aneurisms of considerable degree of development, it has been estimated that half of these cases terminate in rupture of the sac. The statistics of Lemann¹ show that of 592 cases of thoracic aneurism, rupture into nearby structures occurred in the following instances:

	Cases
Pericardium (Fig. 48)	148
Left bronchus, pleura, or lung	160
Right bronchus, pleura, or lung	62
Esophagus (Fig. 81)	50
Trachea	48
Through the skin	35
Superior vena cava	31
Pulmonary artery	18
Other structures	40
	<hr/> 592

Rupture of an aneurism may be a most dramatic incident, especially if it suddenly perforates a bronchus, or it may rupture into other structures and be quite free of symptoms other than those of concealed hemorrhage. I have in mind a patient with clinical symptoms of aneurism of the transverse arch and with a very moderate degree of external tumor formation, who was comfortably propped in bed and pleasantly engaged in conversation. Suddenly her features blanched and her expression became agonized; she clutched at her throat, giving a short cough as though

¹ Lemann: Amer. Jour. Med. Sci., Aug., 1916,



FIG. 81.—RUPTURE OF AN ANEURISM INTO THE ESOPHAGUS.
Cross-section of the trunk below the clavicles, looking from above downward.
(Courtesy of Dr. Elmer H. Funk.)

to clear it of some obstruction, and at that instant huge quantities of blood gushed from her mouth. Life was extinct a few moments afterward.

In contrast with such an incident can be cited the tranquil termination of a patient with an aneurism which ruptured into the esophagus (Fig. 81). "On the morning of his death he seemed in his usual condition, although he stated he felt disinclined to eat. He was sitting on the edge of his bed when he called for the nurse, stating that he felt faint. He was urged to lie down and in about fifteen minutes became quite pale. By the time the resident physician reached his bedside, ten or fifteen minutes later, he was dead."¹

The case of a physician of 33 years who died as the result of rupture of an aortic aneurism into the left innominate vein, as reported by Herrick,² is a graphic description of the emergencies and complex symptoms which may result from aneurismal rupture.

"On the evening of December 9, 1918, he returned late to his office. As he was ascending the stairs he experienced a feeling as of something giving way in his chest; his neck and face felt flushed and full. The sense of choking and pressure was so urgent that as he entered the office he violently tore open his collar and the neckband of his shirt. While trying to turn on the electric light he lost consciousness. How long he lay in a swoon on the floor was not known; he thought about twenty minutes. After regaining his senses he turned on the light, looked in the mirror

¹ From case history notes by Dr. Elmer H. Funk, to whom I am further indebted for Figure No. 81, which illustrates the necropsy findings in the patient.

² Herrick: *Amer. Jour. Med. Sci.*, vol. clviii, No. 6, p. 782.

and saw that his face and neck were greatly swollen and 'almost black in color.' Breathing was difficult.

"The picture presented, as I saw this patient for the first time five weeks after his accident, was one of which I have never seen the duplicate. The neck and face as well as the wall of the chest were swollen as in the anasarca of chronic parenchymatous nephritis; the injected, bulging eyeballs could be seen through the narrow slits left between the swollen lids. But instead of the pasty pallor of the nephritic facies there was a purplish, almost black color, such as is seen only in the most extreme degrees of cyanosis. The visible veins were distended and tortuous. But no feature was more remarkable than the sharp contrast between the bloated, dark, upper half of the body and the pale, emaciated lower portion. The legs were the spindle legs of one in the terminal stages of a wasting disease, with no swelling and no pitting on pressure. There was no sign of free fluid in the scrotal sac or in the abdominal cavity. The liver was just palpable. The abdominal wall itself was not edematous except slightly so above the umbilical level. The line of separation between the non-swollen pale, flaccid abdominal wall and the swollen, purplish chest wall whose skin and subcutaneous tissue felt hard and brawny, pitting only on quite firm pressure, was almost as clear-cut as is the line of demarcation in a case of gangrene, being distinctly marked close to the costal margins.

"Cardiac outlines were difficult to determine because of the thick, edematous chest wall, but the heart was evidently located somewhat to the left. And it was plain that there was an increased area of dullness

at the base of the heart and over the manubrium. Here also could be very clearly heard the murmur already described, which is referred to in the history sheet as follows: 'A systolic murmur which lasts into diastole is heard over the precordium in front and the interscapular region behind. At the aortic cartilage the murmur is particularly distinct and most intense. Here it is continuous, soft, blowing, somewhat humming in character. It is accentuated with the ventricular systole, so that at such times it becomes a loud, slightly roughened blow.' There were numerous râles, both moist and dry, over the lungs, particularly behind. At the bases was dullness with some obscuring of breath sounds as from pleural fluid." Death occurred 8 days afterward. The Wassermann test was strongly positive.

Therefore it is evident, from the statistics and from the instances cited, that the prognosis in an aneurism that is recognized by symptoms or recognized by physical signs is always grave, and the patient's life in constant jeopardy. This statement must stand, despite the fact that we all know patients with well marked physical signs of aneurism who have led lives of semi-invalidism for years.

TREATMENT OF THORACIC ANEURISM.

Manifestly no treatment will restore the integrity of the vessel wall (Fig. 80). Attention should be directed to the relief of symptoms and to the prolongation of life. The time for treatment is well nigh past when the larger aneurisms have formed. The time to have treated the patient was when the symptoms of aortitis or of aneurismal dilatation of the arch of the

aorta presented themselves in months or years gone by.

Should the Wassermann serologic reaction be positive, antisyphilitics are indicated with the hope of minimizing the active inflammation of the vessel wall.

Pain is the most distressing and most persistent feature which one is called upon to combat in aneurism. The routine employment of iodide of potassium in 5- to 20-grain doses three times a day is believed to have some effect in the reduction of pain. Morphine may be required in the dose of one quarter of a grain hypodermically, repeated when needed for further effect. Venesection in the form of occasional small bleedings frequently repeated, is of value in relieving pain and the effect is at times surprisingly long continued. Six ounces of blood may suffice at one venesection. Pain further yields to the introduction of wire in an aneurism, this method of treatment being discussed further on.

Diet.—The dietetic treatment of an aneurism has for its object the thickening of the affected vessel wall by the deposit of coagulated fibrin from the blood. The coagulability of the blood may, to a limited extent, be affected by diet, as may also blood volume and density. For these purposes Tufnell, of Dublin, suggested a rigid diet in aneurism. It is spoken of as "only less rigid than the old method of Valsalva, who gave a half a pound of pudding morning and evening and nothing else—practically starvation!"¹ Tufnell's treatment and diet is as follows:

Absolute rest is strictly enjoined upon the patient; he is not permitted to make exertion of any kind. He should be

¹ Thompson: Practical Dietetics, 3d ed., Appleton, New York.

fed by a nurse. By rest alone the rate of the heart-beat is materially slowed, and this is favored also by the reduced diet; consequently the pressure within the affected artery is lessened.

Breakfast.—Two ounces of bread with a little butter and 2 ounces of milk.

Dinner.—From 2 to 3 ounces of meat without salt and 4 ounces of milk; for a portion of the milk an ounce or 2 of claret may be substituted.

Supper.—The same as the breakfast.

If the patient can be induced to submit to this rigid diet such improvement in the physical signs of an aneurism as diminished pulsation and lessened pain is often marked. Improvement may be looked for within the week. If too long enforced over a period of six weeks, an extreme degree of anemia may develop.

THE WIRING OF AN ANEURISM.

Thoracic aneurisms of the sacculated variety may lend themselves to the beneficial results attendant upon the introduction of a fine platinum-gold wire into the sac, when they are close enough to the chest wall to permit the introduction of the cannula which directs the wire. The purpose of the operation is to prevent a rupture of the sac at that point where rupture is most likely to take place; the wire is introduced into the *sac*—not into the caliber of the vessel—with the hope that a clot may form around the wire and thus retard a rupture of the aneurism wall. It is an operation not to be lightly undertaken. The introduction of the cannula may result in sudden rupture of the aneurism—a possibility that should invariably be explained to the relatives beforehand.

The possibility that the wiring of an aneurism at one point may so deflect the blood stream as to cause a sacculation to appear at another and inaccessible point in the vessel wall, should not deter the physician if the procedure is indicated, the patient and relatives acquiescent, and the case a suitable one.

Preliminary studies of the case are of course made by fluoroscope and by skiagraph. At the conclusion of the wiring the Röntgen light is again requisitioned to determine the position of the wire and establish the success of the procedure.

In the method devised by Corradi, the skin over the aneurism is sterilized and protected from the action of the electric current. From 10 to 15 feet of fine platinum gold wire is introduced through a small porcelain or lacquer-covered cannula. Some aneurisms are of a capacity that will hold 45 feet of the wire. The end of the wire is now connected to the positive electrode of a galvanic battery, and a large wet electrode on the patient's back connected with the negative pole. The current is turned on to 5 milliamperes and increased a like amount every 5 minutes until 50 milliamperes are being used. The acid reaction produced by electrolysis about the gold wire produces a firm clot, and by the end of half an hour pulsation in the sac is often notably diminished. No other alloy than platinum should be employed; a copper alloy will be dissolved under the electric current. If there is an excess of platinum in the wire, it may be so springy as to push aside any fibrin already deposited on the vessel wall, and by its resistance, push out the wall of the sac and thus defeat the purposes of the operation. At the end of an hour the electrodes

are disconnected and the free end of the wire pushed beneath the skin, the cannula withdrawn and the puncture sealed. The procedure has been successful in closing the sac in several instances. One of the most beneficial results is the marked relief from pain, which may occur within 5 minutes following the operation. After wiring an aneurism the patient should remain perfectly quiet in bed for two or three weeks to favor consolidation of the clot.

CHAPTER XXI.

Angina Pectoris.

THE CONDITION DEFINED.

ANGINA pectoris, which literally means “strangling of the breast,” was clinically described by Heberden in 1772. The term is applied to a symptom complex consisting of: (1) paroxysmal attacks of pain, commonly substernal, often radiating down an arm; (2) a sense of constriction within the thorax; (3) a sense of impending death.

It is necessary to enlarge upon the usual conception of angina pectoris, both as to etiology and symptomatology, if one desires to recognize it in the early stages. This condition is capable of early recognition, provided one constantly bears in mind that any one of the three items enumerated in the symptom complex may be lacking—or may be so altered as not to come within the scope of the definition.

The paroxysmal attacks of pain, for example, which have heretofore been spoken of as occurring at the precordium, are far more frequently located under the sternum: indeed they may have their origin along the course of distant arteries and not be felt in the upper region of the thorax at all. Again, while it is perhaps more usual for the pain of angina pectoris to be referred to the left arm, it frequently happens that it is referred to the shoulder, to the neck, to the right arm, to the back or even to the abdomen,

in which latter situation it may be mistaken for a gastric disturbance. In other instances, pain may be altogether absent, as has been recognized from the time of the earliest writers, who defined such a circumstance by employing the term *angina sine dolore*.

The sense of constriction is not invariably confined to the thorax, but may originate elsewhere and be referred to the pectoral region. The *angor*—a term which means great anxiety accompanied by painful constrictions and oppression—is occasionally first felt in the abdomen or along the course of the larger arteries, such as the brachial.

The sense of impending death, which has been usually described as due to fear, is not as a general thing accompanied by a feeling of fear. It has been repeatedly observed that sufferers with angina pectoris are remarkably calm and self controlled when in the throes of an attack, despite the fact that many of them recognize the seriousness of their condition.

It is clear, therefore, that unless one revises the usual conception of angina pectoris sufficiently to include such broader interpretations of symptoms, one may fail to diagnose the condition in its earliest phases.

OBJECTIONABLE TERMS.

It is to be deplored that such terms as “pseudo angina,” “mock angina,” “false angina,” etc., have crept into medical literature. More extended observation of such cases will frequently establish them as atypical instances of true angina pectoris. Time quite generally proves that the “mock” anginas are mild attacks of the true condition, which have been dismissed from consideration and proper treatment un-

der the reassuring diagnosis of "pseudo angina." Ingals¹ who spoke of this subject with a depth of feeling born of intimate acquaintance, stated that the majority of patients with "false" angina die sooner than do those who suffer from diagnosed angina pectoris—the palpable reason being that the former are neglected while the latter receive watchful care and proper treatment.

Hysteria may of course simulate angina pectoris, just as hysteria may simulate any malady. But even though the symptoms prove to be due to an hysterical attack, it is far better for the physician to err on the side of the grave malady and to treat the patient accordingly, rather than to fail to diagnose, or neglect, a condition which time may prove to be angina pectoris.

ETIOLOGY.

Angina pectoris is known as "The disease of doctors," for the reason that it figures so often as a cause of death among medical men, an illustrious example being the eminent John Hunter who suffered with the condition for twenty years and finally died in an attack. High tension living is regarded as a very frequent factor in the production of angina pectoris; this may explain its greater frequency among persons of the better class. It has a much higher incidence in men than in women. While occurring with greatest frequency between the ages of 50 to 70 years, it is not confined to this period of life, but may be observed in young adults and occasionally even in youth.

The etiologic factors may be divided into three great classes; first, the neurotic class which includes

¹ Ingals, E. Fletcher: Chicago Institute of Medicine, Mar. 28, 1918.

persons whose lives are spent under nerve tension and who are given to worry and anxiety. Second, those with a history of previous toxic diseases. Third, those of more advanced years who present other symptoms of arterial degeneration.

Among the infections to which angina pectoris can frequently be attributed are typhoid fever, gout, acute rheumatic fever, and blood impoverishments. The condition is often associated with aneurism, mitral stenosis, pericarditis, and thrombosis. It is probable that the primary infection which induces these cardiovascular affections causes other initial damage which later develops into angina pectoris.

Syphilis is not the all-inclusive factor which some syphilographers would lead us to believe. LeCount¹ has noticed that syphilis is a likely cause in persons under 45 years of age—an opinion in which many observers concur; but considering the comparative infrequency of angina pectoris at this period of life, syphilis is robbed of much of its etiologic significance. Josué² a French physician, believes that angina pectoris is an almost certain sign of syphilis, but was able to secure a positive Wassermann reaction in only 33 per cent. of his cases.

PATHOLOGY OF ANGINA PECTORIS AND ARTERIAL PAIN.

The view that angina pectoris is caused by a spasm of, or by occlusion of, the coronary arteries, thereby depriving the heart of its nourishment, has never proved an altogether satisfactory explanation

¹ Le Count: Jour. Am. Med. Assn., April 6, 1918.

² Josué: Paris Medical Journal, July 5, 1919, 9-27.

of this condition. It has been repeatedly observed at necropsies of persons who expired during an attack of angina pectoris, that the coronary arteries were free from any macroscopic evidence of disease. It has further been observed that angina pectoris cannot be wholly attributed to degeneration of the heart muscle, for persons who have succumbed in attacks of angina pectoris have been found at necropsies to have apparently healthy heart muscle. No evidence of disease could be found. Another hypothesis, which attributes the cause of angina pectoris to sclerotic changes in the walls of the blood-vessels, does not furnish a clinically satisfactory explanation, for the reason that some persons with the most advanced arterial degeneration have no evidence whatever of attacks which simulate angina pectoris.

The views of the English school, which holds that angina pectoris is due to an affection of the walls of the aorta, are much in favor. This satisfactorily explains the clinical phenomena of substernal pain and of constriction within the chest. It does not, however, satisfactorily explain the production of anginal symptoms which begin in parts of the body distant from the thoracic aorta and which later localize, as it were, in the substernal region. It is well recognized, for example, that the first symptom of angina pectoris may be noticed in the left or in the right arm and, as the attack progresses, localize in the pectoral region. Likewise, attacks of pain of the same excruciating and transfixing character as those which characterize angina pectoris may arise in either arm, in the abdomen or indeed in the leg, and the pain remain in these locations and not be referred to the pectoral

region at all. It is a repeated observation of many physicians that attacks of what subsequently proved to be angina pectoris had their *locus* in the abdomen, and were in the beginning confused with attacks of indigestion or other intra-abdominal conditions. Indeed, pain of this nature has been so generally observed that the term *angina abdominalis* has been employed to designate the condition.

What hypothesis, then, can we adopt which will furnish a universally satisfactory explanation for the production of angina pectoris, angina abdominalis or anginal pains which have their *locus* in any part of the body? If we adopt the premises that arteries are capable of producing pain, and that arterial pain is accompanied by a sense of constriction sufficient to inhibit voluntary movement and transfix the sufferer—as any person to whom a tourniquet has been too long applied can testify—a satisfactory hypothesis is forthcoming. It would account for anginal pains far distant from the pectoral region; for “neuralgias” which are not *nerve* pains; for “hysterias” which eventuate in cardiovascular breakdowns; even the pains of intermittent claudication might find an explanation were their etiology thus predicated. What changes could take place in an arterial wall to so pervert its normal function? This subject offers still further material for conjecture. Such a perversion of arterial function might arise from changes in the caliber of the *vasa vasorum* which nourish the media; it might be due to changes in the unstriated muscle fibers which form the muscular coat of the arterial wall; again, it might be due to disturbances in the nerve supply of either the vasomotor apparatus or,

perhaps, to sympathetic nerve affections. We have not yet begun to appreciate the perverted physiologic function which could arise from a disturbance of these delicate structures which enter into the construction of blood-vessels. Future investigation along these lines may reveal a definite explanation of the cause of arterial pain—or the cause may forever remain shrouded in doubt and leave us no firmer footing on which to base the production of *angina pectoris*, *angina abdominalis* and *angina arteritis* than the premises that such anginas are the result of disturbances in a blood-vessel wall.

SYMPTOMS.

The pain and anguish of angina pectoris are succinctly described in Seneca's description of his own case; "the attack is very short and like a storm. To have any other malady is only to be sick; to have this is to be dying."

Pain has its seat in the region which underlies the sternum in those cases where the aorta is involved; but it may be located in other arteries, or may begin in the vessels of the arm or neck and later be referred to the substernal area. Pain is paroxysmal in character, the attacks lasting perhaps only for a few seconds, though they are usually of several minutes duration; they may last half an hour or longer. During the attack of pain there are often momentary intervals in which the pain increases—a waxing and waning of the *angor*, as it were, causing excruciating distress. In a few patients, dull pain may be constantly present for a period of days. In the majority of cases it radiates to the left arm, but it may also

radiate to the right, or involve both arms. It should be remembered that radiation of pain is not essential to the diagnosis of angina pectoris; indeed, as previously stated, there are well marked instances of the condition where pain is not present at all—*angina sine dolore*.

The sense of impending death transfixes the patient to a greater or less degree and inhibits his movements. This sudden arrest of motion which is induced by the *angor* and by the sense of impending death often results in the assumption of dramatic attitudes. For example, when a patient is raising himself from bed his movements may be suddenly arrested by an attack of angina and he be transfixed, resting on one elbow with the other arm thrown out, the chest protruded, the head thrown back, with his gaze fixed on the ceiling. As the anguish lessens the patient slowly assumes a more natural position in bed. The sense of impending death is not due to fear. On the contrary, should a patient attempt to talk during an attack the tone of voice is usually quiet and well restrained. Even though he at times cries out in his anguish, the response to questions between such cries is generally made in a well controlled voice.

Profuse perspiration is to be expected in this anguishing malady, beads of perspiration often standing out upon the forehead. The face is usually blanched and colorless although it may at times be flushed. Dyspnea is not at all characteristic of the condition, but is often present in those cases where the heart muscle is believed to be affected. The pulse, except in the alarm of first attacks, is usually unaltered from its pre-existing condition. If it has exhibited

any irregularities before the attack it is not at all likely to show any alteration in such irregularities, nor even a change in rate, during the progress of the seizure. Any change in rate which does occur is likely to be slowness of the pulse.

It is unusual for the blood-pressure to be at all changed from its pre-existing condition, although in first attacks systolic readings may be elevated. The lungs may at times be acutely emphysematous during an attack of angina pectoris, as described by von Basch.

PHYSICAL SIGNS.

There are absolutely no physical signs characteristic of angina pectoris; those which are present are indicative only of associated conditions and are in no sense diagnostic of angina pectoris. The transverse diameter of the aortic arch may be found increased on percussion and the observation may be confirmed by fluoroscopic examination. An aortic murmur is often present; systolic pulsations due to a dilated aorta may be observed to the right and left of the sternum in the second interspace. The infiltrated blood-vessels of arteriosclerosis are often observed. All of these signs may be absolutely lacking in a given case and, if present, it should be remembered that they are indicative only of associated conditions.

DIAGNOSIS.

The diagnosis of angina pectoris is based upon the symptoms previously described and upon the history of similar attacks. Inasmuch as it is possible for angina pectoris to be preceded by anginal seizures along the course of blood-vessels which are a dis-

tance from the pectoral region, it is well to regard with suspicion the occurrence of *arterial pain*, as being possibly indicative of eventual angina pectoris.

Therefore, if one is to be on the *qui vive* for early symptoms, a sense of constriction after such ordinary causes as fast walking or hill climbing should be regarded as of possible arterial origin. A pain recurring in the same location or under similar circumstances is equally suggestive. Recurrent pain along the course of an artery which is attendant upon the ordinary causes mentioned above, is most significant.

Substernal pain radiating to the left arm or both arms with a feeling of constriction within the thorax and a sense of impending death are definitely diagnostic of angina pectoris.

PROGNOSIS.

Guarded, not necessarily fatal, is the prognosis in angina pectoris. In young subjects or in those in whom it is induced by toxic agents, apparently complete recovery may ensue. Attacks which are brought about by overwork or which arise as a result of high tension living offer, of course, a more hopeful prognosis as to relief than do those which result from degeneration of heart muscle or from arteriosclerosis; under these latter circumstances the prognosis of associated angina pectoris is grave. Patients whose hearts exhibit a fair amount of cardiac reserve force may live through years of successive attacks; on the other hand, the weakened myocardium may succumb early after the establishment of the malady.

Herrick and Nuzum¹ give the average duration of life, in fifty patients who subsequently died with angina pectoris, as nearly three years following the onset of symptoms. It is believed that the immediate cause of death in angina pectoris which terminates suddenly is due to the inception of ventricular fibrillation, although there is as yet no direct proof for this belief.

TREATMENT.

Prophylactic Treatment.—The patient who is subject to attacks of angina pectoris should be cautioned to avoid those causes which precipitate a paroxysm. Attacks may be reduced in frequency in some patients by the avoidance of rapid walking. Walking against the wind is another provocative circumstance. Attacks are invited in many subjects when they attempt to climb a hill or ascend a staircase without pausing at frequent intervals in the ascent. The late James Honan believed that attacks were precipitated in those patients who walked immediately after meals and who were anxious to reach their destination—as for example the business man who hurries to his office immediately after breakfast. Sudden changes of posture, or lifting, reaching or stooping, may also induce the condition. Sudden temperature changes are among the exciting causes. Exposure to cold air, as when the patient is greeted by a wintry blast as he steps from a well heated house, will bring on the symptom-complex in many persons who are subject to angina pectoris. Ingals² expressed the opinion that meteorologic conditions bring on an attack, and

¹ Herrick and Nuzum: Jour. Am. Med. Assn., Jan. 12, 1918.

² Ingals: *Loc. cit.*

mentioned an instance in which a patient who was sitting in a warm railroad car was seized with an attack of angina pectoris when the train ran into a wind storm.

Hurry and excitement undoubtedly favor the occurrence of angina pectoris. Faulty habits of eating, or the ingestion of indigestible foods which cause flatulence, are exciting factors, as is also constipation.

An ocean voyage benefits many patients who are subject to angina pectoris, probably for the reason that they leave care and worry behind and, on board ship, are removed from their accustomed habits of life and find little occasion for the physical effort which, at home, would induce an attack. There are other persons, notably those who are not of the high strung type, in whom ocean voyages produce little benefit.

A further step in the prophylactic treatment of the condition is to search for the cause of an underlying anemia. This anemia may be due to blood dyscrasias, or to focal infections, or to systemic infections, of which latter syphilis is an instance.

Palliative Treatment.—Nitroglycerin is the remedy par excellence in relieving an attack of angina pectoris. When liberally employed it quite generally proves to be an efficient remedy, but when given timidly or in the usual dose of $\frac{1}{100}$ of a grain, but little result can be expected. Ingals stated that he received benefit and noted improvement of symptoms within three minutes after a tablet of nitroglycerin was dissolved on the tongue. The effect of the drug is fleeting, however, and it may be necessary to repeatedly dissolve on the tongue tablets of

the strength of $\frac{1}{100}$ of a grain until the attack is relieved; in this way as much as $\frac{1}{20}$ of a grain may be taken in the course of a day. A violent headache is often the result of administering the drug in unusual dosage. It is, of course, the part of wisdom for the physician to begin with the usual dose of $\frac{1}{100}$ of a grain in a patient whom he sees for the first time, and to gradually increase the frequency of administration until the amount required by the individual patient and the degree of reaction is understood. *Liquor trinitrini*, 1 per cent. solution, given in 10 minim dose, is often efficacious, but requires the lapse of several minutes before its therapeutic action becomes manifest.

Amyl nitrite is employed for relief of an attack by crushing a pearl containing 5 minims in a handkerchief and inhaling the drug. It has an odor which to some persons is sweetish and sickening and is as often disappointing in its effect as it is at other times productive of good results.

Morphine and atropine, in the usual hypodermic dose of $\frac{1}{4}$ grain of the former drug and $\frac{1}{150}$ of the latter, may be used to abate the severity of a prolonged attack. Hot drinks may also be used with benefit in attacks of unusual duration, and external heat may be applied for the purpose of relaxing vasoconstriction.

The use of chloroform to abate an attack of angina pectoris is a dangerous procedure, when there is no previous knowledge of the condition of the heart muscle or of the condition of the arteries (page 409).

Potassium iodide is effective in relieving the anguishing pain of angina pectoris in some instances.

Even though its beneficial effects be not immediately manifest, it very frequently lessens the intensity of a paroxysm and retards the frequency of subsequent attacks. Potassium iodide is not a remedy to be reserved for exhibition in syphilitics alone; it proves effective in cases where there is no evidence of syphilis.

Sodium nitrite in the dose of 1 to 2 grains may be tried in those instances where there be dull, continuous pain. Opium in the dose of one grain morning and evening may also be used under the same circumstances. Carminatives are employed for the relief of flatulence, it being frequently noted by patients that an attack of angina pectoris ceases with belching. It is probable, however, that belching is more often occasioned by air which has been swallowed during the paroxysm, rather than by the flatulence of indigestion.

If the pulse be slow atropine sulphate hypodermically in dose varying from $\frac{1}{250}$ to $\frac{1}{40}$ of a grain a day is approved therapeutics. Atropine paralyzes the peripheral ends of the vagus nerve, and if any part of the attack be due to vagal influence, atropine may correct such influence.

Digitalis.—*Digitalis* is of very little use in the treatment of angina pectoris. It is more apt to produce undesirable results than good effects, and in the opinion of many observers, distinctly aggravates the condition. There is little occasion for its use unless auricular fibrillation coexists with angina pectoris, in which combination of conditions *digitalis* benefits the heart delirium and improves the general circulatory condition of the patient.

Improvement may follow the employment of medicated baths, between paroxysms, in persons who are subject to angina pectoris. The employment of such baths is discussed under Balneotherapy, page 389.

The condition of the heart muscle should be exhaustively investigated in every patient who presents symptoms of angina. The electrocardiograph may at times reveal significant curves that indicate some form of heart muscle involvement. The possibility of Röntgen-ray examination of the mediastinum detecting pathologic conditions should also be borne in mind.

CHAPTER XXII.

Neuro-circulatory Asthenia.

THE NAME.

DA COSTA, writing in 1871 of certain nerve and circulatory phenomena which he encountered among military men of the Civil War, employed the term "irritable heart of soldiers." Early in the recent World War such cases were referred to by military surgeons under the phrase "D.A.H."—disordered action of the heart. Inasmuch as the characteristic symptoms of the condition—hurried respirations, rapid heart rate, precordial pain, tremulous fingers and bluish hands were *disproportionate to the slight amount of physical effort required to call them forth*, English writers later employed the terminology "effort syndrome" to describe the condition. A still further refinement of nomenclature, *neuro-circulatory asthenia*, seems to be an apt phrase to define the asthenic who is a potential cardiopath and a potential neuropath as well. Perhaps the limit of terminology for the symptom-complex has not yet been reached; a generation later than ours may credit the one who first described it and give it such a name as "DaCosta's circulatory syndrome."

THE SYNDROME IN CIVIL LIFE.

Neuro-circulatory asthenia is not at all peculiar to military life. It exists, active or quiescent, among

civilians of all classes of society. It is typified in the rather frail, neurotic, emotional person who collapses in psychic emergency or who breaks down under trifling physical strain. The hurried respirations, rapid heart-rate, precordial pain, tremulous fingers and bluish hands of this type have caused them to be referred to in the past as having an "excitable heart," a "nervous heart" an "adolescent heart;" the condition has been designated "functional heart disease," and even referred to by the unpardonable term of "masturbator's heart." There are lesser degrees than this extreme type in civil life, found among people of sedentary existence—occasionally, too, among those of slothful habits—whose nervous and circulatory systems are quite sufficient to the demands of their work-a-day life, and to any additional burdens incident thereto.

THE SYNDROME IN TRAINING CAMPS.

Now, let such an under-developed, yet self-contained and efficient individual, be thrown suddenly into the military whorl, with all its rigors and strains. Submit him to the unusual physical demands thus imposed on undeveloped muscles; deny him his accustomed periods of rest; bend the individual's personal inclinations to the will of a distasteful or overbearing military superior; subject him to the constant fear of court-martial for some slight and unintentional infraction of army rule; surround him with the taunts of jibing comrades that prod him on when wearied nature demands rest for exhausted mind and body—it would seem that there could be nothing in the world that would more quickly make a neuro-circu-

latory weakling out of a constitutionally inferior person than the army gaff. Perhaps this is the reason more of these cases are observed in training camps than in civil life. At this stage the asthenic can still be saved to society if spared further effort; he can be reconstructed, if painstakingly treated in development battalions.

THE SYNDROME IN WAR.

Take one with the above initial conditions and add the physical drain and the infections often attendant upon exposure, lack of sleep and poor nutrition on the battle-field; add, if you please, the shock of conflict and the carnage of war: and it is then found that one who was a perfectly efficient producer at home has become a quite useless waste-product of the front, fit to serve in neither civil nor military life. Such is the evolution of the neuro-circulatory asthenic in war.

PREDISPOSING CONDITIONS.

Although neuro-circulatory asthenia arises for the most part in individuals of the neurotic, neurasthenic or neuropathic type, who also have family histories along these lines, it is not necessarily confined to this group. Tuberculosis predisposes to the syndrome. So does goiter—so do low-grade infections, such as trench fever. It may arise if physical activities interrupt an insufficient convalescent period from pneumonia, typhoid fever, or influenza. It may arise as a result of the inhalation of poisonous gases that affect the heart as well as lungs and bodily metabolism. In short, *any condition that renders one constitutionally inferior* furnishes the canvas upon which the clinical picture of the *effort* syndrome may be painted.

TYPICAL CASE RECORD.

For the purposes of this discussion I have analyzed a group of 100 consecutive cases of neuro-circulatory asthenia. From this analysis a typical case-history can be constructed, as follows:

X. Y. Z., Age, 25 years. White. American. Clerk. 5 feet 6 inches. 135 pounds.

General appearance: Healthy, frail. Hands bluish cast.

Development: Slender.

Skin: Pallid, fine texture.

Family history: Mother subject to "nervous breakdowns"; had "spells with her heart." Brother of slender build. Sister "nervous."

Previous diseases: Irrelevant.

Habits: Irrelevant.

Personal history: Never indulged much in athletic games. Tires easily on physical effort. Has stopped work and taken to bed for brief intervals on occasions in past two years, on account of "having worked too hard" and being "nervous." Gave up job requiring physical effort and took clerking position.

Presenting symptoms: Pain over heart, shortness of breath and palpitation following exercise. Easily excited.

Physical examination: Thyroid gland palpable. Fine tremor of finger tips. Precordial hyperesthesia. Arteries elastic, pulse of usual volume, regular, rapid. Precordial impulse diffuse, striking, quick. Apical systolic thrill when erect.

Cardiac borders: Transverse 12 cm., apparently not increased after exercise. Maximum cardiac impulse $7\frac{1}{2}$ cm. from mid-sternal line.

Murmurs: Apical systolic, not transmitted; P₂+

RATES	Before Exercise	Immediately after 100 hops	Two minutes later
Respiratory	16	34	24
Ventricular	117	151	127
Radial	117	151	127
Deficit?	0	0	0
		Moderate dyspnea	

Diagnosis: Neuro-circulatory asthenia, moderately severe.

FURTHER POINTS IN ANALYSIS.

It is interesting to carry the analysis of the hundred cases a bit further, as it gives one a more elastic picture of neuro-circulatory asthenia.

Reasons Referred.—Forty per cent. were referred for tachycardia; twenty-six per cent. for mitral disease. The latter diagnosis was probably based upon the detection of apical systolic murmurs, as they were present in 26 hearts. (Valvular disease did not exist in any of the patients). Twenty-two were referred with the sentence "Heart examination desired," showing that their hearts had been recognized as departing, in some indefinite manner, from usual standards.

Previous Diseases.—Each man of the hundred had one or more of the following diseases earlier in life.

Abscessed ears	7	Pertussis	53
Abscessed teeth	12	Pneumonia	17
Chorea	1	Rheumatic fever	3
Diphtheria	13	Scarlatina	9
Gonorrhea	18	Syphilis	3
Measles	74	Tonsillitis	24
Mumps	55	Typhoid fever	11

Habits.—Seventy-one used tobacco five or more times a day; 32 smoked 5 to 10 times a day, 39 ten or more a day. Inasmuch as a previous investigation (see page 38) had shown that 87 per cent. of a thousand American male adults smoked on an average of 11 times a day, there seems to be no fundamental connection between the tobacco habit and neuro-circulatory asthenia. As to alcohol, 35 were accustomed to imbibing an ounce or more of spirits every 24 hours.

Presenting Symptoms.—Fifty-four of the hundred cases complained of precordial pain, especially after

slight exertion. The following complaints had been present for an average period of $2\frac{1}{2}$ years: Giddiness in 67; palpitation in 63; shortness of breath in 38 and fainting in 20 instances. Forty had, on various occasions during that time, found it necessary to stop work and take to bed as a result of these symptoms.

General Appearance.—Forty-nine were of the usual healthy facial appearance; 23 were pallid; 20 were florid. Fifty-four had cyanosis of their hands and forearms in varying degree, especially noticeable when the hands hung dependent. Fifty-four showed tremors of the finger tips of the outstretched hands before exercise, or else tremors of the lightly-closed eyelids.

Concerning the mental attitude which these neuro-circulatory asthenics assumed toward their cardiovascular examination: fifty-four displayed no more than the usual interest in the maneuvers; 25 were alert; 11 were anxious; 10 were apathetic.

Thyroid Gland.—Of the hundred, 68 had palpable thyroid glands, varying in degree from those which had to be carefully sought for by palpation, to most apparent thyroid enlargements.

Pulse and Pulsations.—The pulse in the greater number of cases was regular and of good volume. Sinus arrhythmia was present in 12 of the patients. The arteries, for the greater part, were of the usual elasticity found in health. Visible pulsations were not observed in the neck, in the brachials or at the base of the heart; at the apex a forcible impulse was present in 37 of the cases. The precordial impulse was diffuse—"irradiation"—in 60 of the hundred.

Blood-pressure Estimates.—The systolic estimates were quite what one would expect to find in neurasthenia—never constant, often low—frequently elevated, perhaps 20 points over what is considered usual.

Pulse Rates.—The average pulse-rate was 117 before exercise; it was 151 immediately after 100 hops, and 127 two minutes following. These figures do not represent the full excursus of the neuro-circulatory asthenic pulse. In some the rate was 84 before exercise; in others it shot up to 204 immediately after. An occasional patient was not exercised at all, as it was felt that an initial pulse-rate of 144 was sufficient warrant for not adding to the load of a tumultuous heart in seven of the 100 patients. Eight were unable to finish the test on account of urgent dyspnea arising. Two collapsed completely and fell to the floor with air hunger.

Cardiac Borders.—The right border was believed to average 2.4 cm. from the midsternal line; the left border averaged 9.5 cm. The maximum cardiac impulse averaged 7.7 cm. from the midsternal line, in the fifth interspace. There is nothing unusual in the measurements. It was interesting to note that in not one of the cases did palpation and percussion reveal an increase in the left border following exercise.

Heart Sounds.—Murmurs were present, principally at the apex, in 32 instances. An apical systolic murmur was heard in 26 cases and was transmitted to the axilla in seven of that number. The pulmonic second sound was accented in 45 of the hundred cases. The aortic second was accented in 3. Among the 68 cases in which no definite murmur was heard at

the mitral area, 34 had a marked alteration in the character of the mitral first sound, described as *snappy* in 13 of the hearts, as *prolonged* in 14 and as *plus* in 7. In other words, there were deviations in the auscultatory phenomena in every one of a hundred cases.

TREATMENT.

Going back in memory a few years, one can recall the "marvellous heart cures" that returned to America, boasting of the wonderful results of treatment at German spas. Some patients had, unquestionably, been benefited—and now one asks one's self the question: were not these "cured" cardiopaths of that time similiar to the neuro-circulatory asthenics of today? Spa treatment certainly combines all of the essentials that contribute to the restoration of the neuro-circulatory asthenic. Freedom from accustomed worries and from the routine cares of a too-familiar daily life; the avoidance of physical strain; quiet, peaceful surroundings; the interest of new acquaintances; regulated daily habits; personal hygiene; skilled nursing, but no others than the first few days spent in bed; dietetic regime; elimination; the soothing effects of baths; graduated exercises. Perhaps this treatment explains why more than one "enormously dilated heart"—as many a neuro-circulatory asthenic believes himself to have when he notices his diffuse precordial impulse—returned to normal size! Be that as it may: the treatment which the "cured" cardiopath received is the ideal treatment for the neuro-circulatory asthenic of today. (The adaptation of graded exercises is discussed on page 383.)

CONCLUSION.

The question is frequently asked—"Which system, nervous or circulatory, is *primarily* at fault in neuro-circulatory asthenia?" Neither *is*, necessarily, but either *may* be. The syndrome arises as a *result of continued or repeated emotional strain or physical stress in excess of the capabilities of a weakened or constitutionally inferior person*. He may be congenitally weak, or he may be weakened by accident, by physical strain, by emotional stress or by disease; the weakness may be in nerves, or circulation, or lungs, or glands, or muscles, or bones. Wherever the weakness be, the nervous and circulatory systems endeavor to maintain the individual's *physiologic balance*. Their efforts in this direction may be no greater than those put forth by other bodily functions in the same common cause, *the struggle for physiologic balance*. But it happens that the nervous and circulatory systems are noticed more than the others, for theirs is the louder voice of complaint.

CHAPTER XXIII.

“What Can be Done for Heart Disease?”

THERE is a general impression, not altogether confined to the mind of the laity, that little can be done for heart conditions after once establishing the diagnosis. Indeed it is not unusual to hear the remark that all one can do for a person suffering from a heart affection is to put the patient at rest in bed and administer digitalis!

Consider the case of tuberculosis. Even the younger physicians of this generation will have no difficulty in recalling the time when a diagnosis of pulmonary tuberculosis was quite equivalent to the pronouncement of a death sentence upon the patient. Despite that gloomy prognosis, which was nurtured in the mind of the public by the skepticism of the physician, there were many excellent students of medicine who devoted their lives to the recognition of early signs and to the development of rational treatment for what was then called the “great white plague.” The discovery by Koch, in 1882, of the causative bacillus gave an impetus to the studies of tuberculosis, and the results since achieved by the employment of proper feeding, elimination, hygienic and sanitary measures, are a triumphant vindication of those men of vision who insisted, in spite of popular opinion, that tuberculosis is a preventable and curable disease. The education of the public to this belief has resulted in the building of vast sanatoria with public

funds, where such patients can be segregated and skillfully treated. He would be rash who, in the present day, would utter in the presence of even the most uneducated layman, a hopeless prognosis in every case of pulmonary tuberculosis. Such is the change that one generation has seen in the treatment of a chronic lung affection.

The tuberculosis situation of a generation past finds a parallel in the study of heart disease today. From the time of Celsus and Galen there have been philosophers who believed that the treatment of heart disease could be put upon a rational basis. Despite the skepticism which surrounded them, investigators such as Morgagni, Corvisart, Laennec, Hope; Stokes, Corrigan, Quain, Balfour, and Babcock persisted in the study of cardiac conditions and evolved methods of treatment which bore fruit in their day, and which form the basis for modern therapeutic measures. Heart disease, it is true, has no specific bacillus for a present day Koch to discover; but Mackenzie, the Father of Modern Cardiology, has performed an equivalent service by devoting his life to the pioneer work of identifying by means of graphic records, early cardiac conditions which may be fore-runners of extensive heart damage. It will probably never be necessary for the state to maintain institutions for the segregation of patients with heart affections, for heart disease, *per se*, is not communicable: but in the large centers of population cardiac clinics are already springing into existence at a gratifying rate and have for their purpose the treatment and *instruction in self-care* of persons so afflicted. The education of the public should be continued until the

earlier symptoms of heart affections are so generally recognized that patients will, of their own accord, consult their physicians at a time when beginning cardiac defects can be prevented from developing into the hopeless heart wrecks that stigmatize our profession. Indeed, while it may seem Utopian, such instruction should be carried to that point where a person who has suffered from *any* acute infection will appreciate the necessity of a thorough cardiovascular examination before venturing a return to his usual manner of living, and have the examination repeated a few weeks after he has entered thereon. This instruction of the public must come from the physician; and if the *Doctor in Arte Medica* is to be a true "Teacher in the Art of Medicine," he must forever abandon the skepticism which now belongs to the past and harmonize his mental attitude with the trend of the professional thought of today, which teaches that *heart affections belong in the category of preventable and curable conditions*; but as in the case of tuberculosis, both preventability and curability depend upon the recognition of *early symptoms*.

THE EARLY RECOGNITION OF HEART DISEASE.

Ten years ago, in many quarters, a diagnosis of heart disease was built upon gross physical signs. The symptoms of which the patient actively complained were accorded little significance, so long as his heart was free from murmurs, his legs free from swelling, his respirations free from labor and his features free from cyanosis.

In those days, when an apparently healthy person

presented no symptoms other than shortness of breath following accustomed effort, pain in the chest, palpitation, or a feeling of faintness, or when he asserted that his heart "turned over" at times, it was customary to listen at the precordium and reassure him with the statement that perhaps he had "a little digestive disturbance," or else that his heart trouble might be "functional, certainly not organic." *Functional!* A functional fault in a heart could be lightly dismissed—but suppose it had been his leg that was not functioning properly; would not a more thorough search have been made for the cause?

Today, however, the profession of medicine is awakening to the necessity of adopting finer diagnostic technique and newer methods whereby heart disease can be recognized in its incipency. Nowadays, when a healthy-looking patient complains of shortness of breath, precordial pain, palpitation and a feeling of faintness he has, first of all, a thorough search made into his history, habits and occupation—for past or present diseases, past or present habits, past or present occupation, may influence the heart in divers ways which this volume has endeavored to set forth. The individual's pulse is carefully studied and any variations in rate, rhythm or volume noted. His heart borders are outlined to determine significant increases in diameters and, if need be, these clinical observations are supplemented by Röntgen ray examination. He is put through an exercise test to determine the rate-response of the heart to effort. A polygram may be required to elucidate a vague arrhythmia, or an electrocardiogram be demanded in order to clear up a doubtful diagnosis. By such an

examination does the physician secure the testimony which enables him to diagnose an early heart affection before it advances to the stage of cardiac bankruptcy.



FIG. 82.—FOREIGN BODY IN THE HEART.

Sharp eyes will be able to discern the needle to which the arrow on the reader's left directly points. The arrow on the right points to the inflammation of the myocardium where the end of the needle impinged at each pulsation of the heart. This heart is from a former patient of the Norristown asylum. Supposed duration, over two years. (Courtesy of *Dr. Allen J. Smith.*)

WHAT CANNOT BE DONE.

The modern school of pulmonary teaching makes no claim that destroyed tissue can be replaced with healthy lung; nor does the modern school of cardiology assert that heart structure altered by disease is

capable of regeneration. Valve leaflets that are distorted by scar tissue can never again be made to approximate each other. Heart muscle structurally altered cannot be made to regenerate itself. There is no alchemist's art for the heart. Nothing can be hoped for as concerns the reconstruction of tissue. Surgery which has come so notably to the relief of humanity in the early treatment of cancer, affords no hope in the treatment of heart conditions except in an indirect manner, as by operations on septic foci. Surgical manipulation of the heart itself is of such infrequent occurrence and so dependent upon the happy accidents of chance as to constitute surgical dramatics. The premises enumerated above are obvious, but it is fatuous to use them as a foundation for therapeutic nihilism, or for assertions that "nothing can be done for heart disease." Such specious utterances rob the cardiac sufferer of the hope that should be his, and paralyze constructive thought on the part of the medical attendant.

WHAT MAY BE DONE.

For convenience in studying, the treatment of heart affections can be divided into three general procedures, namely: (A) *prevention*, (B) *correction* and (C) *conservation*, which may now be considered in the order named.

PREVENTION.

The Infections of Childhood.—The prophylaxis of heart affections resolves itself, for the greater part, into watchful care of the heart during the course of acute infections of childhood and adolescence. Chil-

dren who suffer from "the usual diseases of childhood" should be the subjects of careful heart-watching during the progress of such hitherto thought harmless, now believed pernicious, infections. For it is then that the *initial* damage, which perhaps does not openly reveal itself for years, may be imposed upon the heart. Little sufferers with even the milder forms of childhood diseases should not be permitted to play about the house during the progress of the infection; they should be kept in bed in order that heart effort may be conserved. A quarantine period, as established by law, protects the heart to some degree by restraining the child's natural incentive to frolic with its comrades; but this period is usually not sufficient for thorough convalescence.

The child may be fretful, restless and difficult to control during the period of enforced rest in bed or indoors; to restrain it without the employment of threats or an exhibition of force often calls for the greatest degree of tact on the part of the nurse and for sublime patience on the part of the mother. The purpose of restraint is to relieve the heart of unnecessary effort; fretfulness, or fear accelerate the heart rate and defeat this purpose. Many other little sufferers, however, seem to realize the seriousness of their situation and will submit to control with a resignation that at times is pathetic. The tedious hours of convalescence should be whiled away with games, puzzle pictures, building blocks, picture books, drawing and painting materials; and when other entertainment palls, few indeed are the children who are not content when an adult reads from a nursery book or tells a story. Other children should not

visit the little patient even though the danger of contagion be past; for playmates, by telling of their games or of their plans may sow the seeds of discontent and unrest in a mind that was perfectly satisfied with the attentions and diversions of care-taking elders. No time-limit can be set on the duration of convalescence from the acute infections of childhood; it is gauged by a study of the individual patient and by the child's return to a usual state of health. The word "convalescence" means "to grow strong," and it is infinitely better to err on the side of conservatism rather than to impose the burden of premature exertion upon a weakened heart that has not yet "grown strong."

During convalescence and in the activities following return to play the little patient should be frequently examined to determine whether pulse, rate-response and early fatigue bespeak a myocardial aftermath. If electrocardiography be available it should be employed in the study of the child's heart; and it should show normal curves before heart vigilance is relaxed, following the acute infections of childhood.

Prophylaxis embraces the much-neglected point of *sufficient period for convalescence*, for adults as well as for children. An adult who has weathered the storms of acute rheumatic fever, chorea, the more virulent attacks of tonsillitis, puerperal sepsis or other septicemia, should of course receive instruction from the physician that the convalescent period is to be not a matter of days but weeks. Yet even a greater danger to the heart lies in the neglect of heart care following *minor* infections, the cardiac

significance of which is lately being recognized. For example, influenza is an increasingly common cause of heart maladies—not for the reason that the heart itself is affected during the short period of infection, but for the reason that patients who recover from influenza frequently undertake their customary duties without a sufficient period of convalescence. The effort which physical fatigue entails upon the heart—the demands which the organism makes upon heart muscle in order that fatigued bodily tissues may be sustained—results in a depletion of cardiac reserve force and thus lays the foundation for cardiac exhaustion, and perhaps for direct invasion of heart tissue by germs. It would be a safe rule for the family physician to insist that a patient who has had *any* acute infection should not be permitted to permanently leave bed until the rate and rhythm of the pulse show no marked variation on attempted effort; nor even after getting up should the patient be permitted to resume accustomed activities so long as a sense of physical exhaustion and an increased heart rate are manifest at the close of a convalescing day.

Prophylaxis of heart disease finds a further employment in the search for constitutional conditions such as occult tuberculosis, latent syphilis, thyrotoxicosis; or for focal infections, such as arthritides, carious denture, intestinal autointoxication—which may impair the efficiency of the individual and throw a load upon the circulatory apparatus. Especially should a search for a systemic condition, which may be causing a cardiovascular affection, be diligently prosecuted when the patient complains vaguely and indefinitely, perhaps, of substernal pain, precordial

distress, dyspnea on moderate exertion or an inability to perform accustomed tasks without distress.

Under prophylaxis should also be considered the habits of the patient, many of which are known to be factors in cardiac derangement. Among these may be mentioned, driving the human machinery at high tension; the pursuit of business or pleasure far into the night; pernicious dietetic habits; over-indulgence in physical exercise; hurry, worry, care and anxiety; and the habitual use of drugs. Here can be mentioned, too, the unnecessary and unwarranted exposure to infections which immoral habits engender; clean living in youth often brings as its reward a heart that is quite sufficient for ripe and mellow old age.

The Patient's Daily Life.

Patients who have recovered from acute infections, especially if there was evidence of heart involvement at the time, often require elemental instruction as to the intimate details of their everyday life if they are to avoid the risk of subsequent heart strain. In briefly reviewing the subjects upon which patients seek advice, the remarks will be made to apply not only to the prevention of heart conditions, but also to the correction and conservation of hearts already affected.

Bathing.—The daily bath which is taken on arising may be either a tepid sponge or a tepid tub bath. The temperature of the water should be between 92° and 94° Fahrenheit. Prolonged immersion of the body is to be avoided. A warm towel should be used briskly for drying the body, and the bather

should rest on bed or couch for a few minutes following a bath.

Cold water baths, whether sponge, shower, tub or sea, entail a degree of shock and reaction to which only robust persons should be subjected. Hot baths are very relaxing and not advisable for cardiopaths. Superheated cabinets, Turkish or Russian baths are usually too exhausting to risk their employment.

Bathing should, of course, be avoided immediately after eating or when digestion is in progress.

Clothing.—Woolen underclothing may be required in rigorous climates. As a general proposition, however, it irritates the skin of the wearer and is a poor absorbent and distributor of bodily moisture. Cotton underwear is more desirable.

There is a tendency on the part of semi-invalids to burden the body with clothing which by its bulk, hampers free bodily movements and imposes unnecessary weight. There are other persons who, following the dictates of fashion, appear in unseasonable weather with neck and arms exposed, wearing stockings of thin silk with low cut shoes. One manner of dress is as extreme as the other. The purpose of clothing is to protect the body and keep it warm and comfortable according to season. Too much clothing, by hindering free muscular play and by keeping the body damp with perspiration that is perhaps barely perceptible, is as much to be condemned as is over-exposure of parts of the body where the cooling of the surface circulation cannot help but reduce the powers of resistance of the individual.

Effort.—The rapid mounting of stairs should be avoided. It should be customary with physicians to

advise convalescent patients that they may climb the stairs but once a day, pausing for a moment's rest in both ascent and descent. Hasty movements of all kinds should be forbidden. The change from a recumbent to an erect position should always be deliberate and guarded. Shortness of breath following any attempted effort is a signal for the prompt cessation of effort. Stooping, as in caring for a furnace, and reaching, as in fumbling for an overhead light, frequently precipitate dizziness or vertigo. The lifting of articles such as chairs or coal pails, or the carrying of loads or bulky packages strains damaged heart muscle unnecessarily. Ploughing through snow or laboriously picking the way on slippery surfaces, have been responsible for many prostrations in cardiopaths. Bursts of speed are utter folly for the convalescent and may retard such a person for weeks in his progress to strength. Affected arteries may respond to haste with sudden attacks of severe pain; while the result of bursts of speed upon affected heart muscle is too well illustrated by sudden death in those who hurry for trains to require further mention.

Exercise, including walking, is discussed farther on in this chapter as a therapeutic measure. Walking may be mentioned here in connection with the advice which the physician should give a convalescent patient concerning walking in the wind, which imposes effort on even the strongest hearts. Talking when walking is also an effort that is poorly borne by persons who are constitutionally delicate. Exercise at times is distinctly contraindicated. There are many people who have arrived at a time of life when the

wear and tear of years is beginning to tell on the circulatory system; and because they have always indulged in brisk walks they still insist on doing so, despite the fact that exhaustion, rather than the customary exhilaration, is the consequence. Absolute physical rest for a short period is the indication in such persons—after which short excursions of gradually increasing distances may be resumed.

An afternoon rest of an hour or two is always advisable for those who are returning to health. The siesta should be preferably taken after the mid-day meal and amid quiet, sleep-inducing surroundings. Clothing which interferes with perfect physical relaxation should be removed and the room darkened, as far as is compatible with ventilation, before the person lies down to rest.

Social affairs have an element of value in that they stimulate the desire of the patient to mingle with others and thus take the mind off of a recent illness and prevent the convalescent from cultivating a too-introspective frame of mind which in many borders upon hypochondriasis. The well mind concerns itself, for the most part, with three subjects—persons, things or ideas; the minds of ailing persons are quite generally concerned in a large degree, with their own physical condition. To be brought in contact with normally-acting minds is a therapeutic measure of no small benefit in the creation of a healthy mental attitude in the semi-invalid. Social affairs should of course not be continued into the hours intended for rest; receptions, card-parties or theatres that entail physical effort or that strain the endurance of the individual should not be attended.

Early to bed should be the rule of a convalescent. Individuals vary as to the number of hours of sleep required. It is said that Edison rarely spends over four of the 24 hours in bed; there are other persons who are fatigued for the day if they have less than nine hours sleep. Despite such individual variations, a person who wishes to exercise the greatest care of his circulatory system will relieve it of the effort of the erect posture for at least eight hours and for as many more as a sense of weariness or fatigue demands.

Many people fall into pernicious habits of thought at bedtime by reviewing the day's events or by planning the morrow. Others indulge in brain work until the mind is so alert that slumber is elusive. Still again there are those who devote the evening hours to exciting games or to forms of exercise that scatter repose—and there is also the unhappy class who discourage sleep by brooding over past or future fears. One should be mentally passive at bedtime. All forms of physical or mental employment should cease an hour before retiring.

Sports.—Fishing, on first thought, would seem to be a form of sport of which even the infirm could freely partake—but it is to be remembered that the art of angling is divided into still and active fishing, fresh and salt water fishing. Still fishing from the shore or boat is an excellent pastime, with a day in the open, moderate exercise and the stimulating element of chance to commend it; the cautious and guarded movements in which an experienced disciple of Walton indulges rather than frighten a possible fish that might be in the neighborhood, are well within the

range of physical effort of which damaged hearts are capable. Active fishing, however, such as trudging along a trout stream, through underbrush and over boulders, with the constrained positions that are sometimes necessary in casting a fly with accuracy, impose a burden on easily exhausted hearts. I have seen more than one lawyer, stale from a winter in the court room, collapse after a happy day along a mountain stream.

Salt water fishing is unobjectionable for those who enjoy this form of sport, providing a companion is along to relieve the cardiopath of the physical strain of landing a heavy fish which interposes a gamey resistance to the line.

Rowing is healthful exercise for the cardiopath, providing it is judiciously indulged in. The heading of a boat against the wind or pulling it in the face of a storm; the loading of the boat with passengers whose weight adds to the effort of the stroke; rowing in contests or boating in a broiling sun should all, of course, be interdicted.

Swimming should be advised against. It has the objectionable features of the initial cold plunge, the overcoming of the resistance of the water and the simultaneous employment of leg and shoulder muscles which precipitate sudden exhaustion. One may speculate as to how many drownings of "expert swimmers" are due to precipitate cardiac exhaustion or arterial spasm.

Skating is an exhilarating pastime whether it be on ice or floor. There is no reason why it should not be indulged in by a young person who is accustomed

to this form of sport and who has apparently recovered from a heart affection.

Golf is a form of outdoor sport particularly suited to the confirmed cardiopath and to those with the arterial changes of advancing years. However, moderation should be used and in some cases it is wise to play only a few holes, rather than to force oneself around the entire course, thereby inducing exhaustion.

Cycling at moderate speed, on level surfaces and along dustfree highways is quite unobjectionable.

Dancing once or twice in the course of an evening is permissible for an ex-cardiac patient, but dancing should not be carried to the point of physical exhaustion and should be discontinued upon the first appearance of shortness of breath, dizziness or fatigue.

Tennis, ball-playing, boxing and wrestling all have elements that make them undesirable forms of sport for any but the constitutionally robust. All are more or less violent; all necessitate stooping and reaching; the possibility of disabling accidents is always present in all but tennis; and in each one there is *the element of contest and desire for supremacy* which so often tempt an otherwise thoughtful person beyond the point of fatigue into actual physical exhaustion. When advising a patient as to permissible forms of exercise or of sports, it should be borne in mind that, as a general rule, *solitary* sports or games are likely to prove more beneficial and are certainly less fraught with undesirable possibilities than are those in which the *element of contest* enters.

Motoring imposes a strain upon the muscular and nervous systems of those who drive their own cars

in city traffic or along unfamiliar highways. For those in the tonneau motoring has an exhilaration, but this can be pushed to extremes and a cardiopath be relaxed and distressed if the drive has been too long continued.

Horseback riding may be permissible for those patients who are accustomed to it and who do not ride furiously. Persons not accustomed to riding horseback should not adopt it as a convalescent exercise. It requires skill and experience to manage a horse, which is often capricious and which interposes a brute resistance to the will of the rider, and thus makes a burden of what is otherwise a healthful form of exercise.

Climate.—It is beyond the scope of this chapter to enter into a discussion of various climates and their indications. It may be generally stated that the desirable climate for a person with a cardiac condition is one that has a mild, dry, bracing atmosphere. Sudden temperature changes should be avoided. Plenty of sunshine is conducive to outdoor exercise. Some people do well at sea level, other at elevations. There is no way of telling in advance which will give the patient greater benefit; the preference of the individual should be consulted when first making the choice between mountains and sea. Altitudes that are over three thousand feet are not to be advised for cardiac patients.

Marriage.—The physician is often consulted concerning the expediency of marriage of a person who has had a heart affection. The psychic value of companionship and of a renewed interest in living are not to be lightly passed over in considering the question.

Unquestionably those suffering from a chronic systemic disease or from a gradually progressive affection of the heart muscle or arteries—as illustrated by a progressing myocardial lesion or by an aneurism—should be advised against entering into marriage. Valve lesions, *per se*, do not contraindicate matrimony, providing the associated myocardial change has been arrested. Persons who have recovered from an acute heart affection and who have enjoyed apparently normal good health without any relapses for a period of a year or two can enter upon matrimony with the caution that any exhaustion of their physical reserve force may entail another cardiac siege. Those who have “nervous hearts,” or the recovered neuro-circulatory asthenics, are frequently distinctly benefited by marriage. Middle aged persons with chronic myocardial change may enter upon matrimony providing they are not subject to periods of circulatory failure; in such a case a physician will scarcely advise that the semi-invalid marry. A word of caution should be given those elderly persons with the arterial degeneration of advancing years who contemplate marriage—especially if with one many years their junior.

It is not indelicate to remark in this connection that persons who have had cardiac affections should enter cautiously upon the intimate relations of marriage. A lack of restraint or overindulgence in sexual gratification may precipitate acute heart symptoms. Especially is this so in people who are subject to periods of tachycardia which is of undemonstrable cause, in chronic fibrillators and in persons with heart-block of any degree. A silver-haired roué of irrepressible personal magnetism, who suffered from

heart-block (Fig. 27, page 99, is his tracing), informed the writer that his amative proclivities were usually interrupted by a complete loss of consciousness, which was preceded by the sound of a pistol-shot in his brain—but his accustomed companion administered "restoratives" and bathed his head with ice water until returned consciousness and increased pulse-rate enabled him to complete the act, even though completion meant 2 or 3 weeks in a hospital recovering, to a degree, from symptoms of acute myocardial failure.

Pregnancy is not fraught with danger to the one-time cardiopath who has apparently recovered from a heart condition and who has not been subject to periods of relapse. Even in persons with chronic valvular lesions which are evidently not progressive, it is remarkable how the heart will sometimes meet the demands which pregnancy and childbirth throw upon it; this heart response, however, is a question of cardiac muscle reserve force, and heart patients should be carefully watched for symptoms of approaching myocardial failure as pregnancy progresses.

CORRECTION OF PERVERTED FUNCTION.

Under this head may be included minor affections and disturbances of the heart which can frequently be relieved by the removal of the cause, and thus be prevented from developing into more serious cardiac maladies. Among such causes which are capable of correction are irritable gastric conditions, which may pervert the action of the heart, thus disturbing its volume, rhythm or regularity; the heart becomes efficient again when the gastric condition yields to treat-

ment. Gall-bladder disease or chronic suppuration of the middle ear are examples of focal infections which may be reflected in cardiac embarrassment, which embarrassment disappears upon removal of the cause (other focal infections are discussed on page 224).

The irritable heart of neurasthenia is also capable of correction. Neuro-circulatory asthenia is a combination of nerve and circulatory phenomena, both of which are often relieved by correction of the underlying systemic weakness. The brilliant results which can also be secured in the correction of such perversions of heart function as auricular fibrillation, auricular flutter, and paroxysmal tachycardia of ventricular origin, are examples of what one may accomplish in the correction of perverted function.

CONSERVATION.

The conservation of heart or blood-vessels which have been irreparably damaged by disease or neglect includes a variety of measures which, employed either singly or in combination, will enable the physician to add to the physical comfort and perhaps to the tenure of life of the persons so afflicted. Among such therapeutic resources are:

1. Rest.
2. Exercise.
3. Diet.
4. Massage.
5. Sanatorium treatment.
6. Operative procedures.
7. Balneotherapy.
8. Cardiac drugs.

These measures, however, are useful not only in the conservation of badly damaged hearts, but also in

the correction of perverted function and as prophylactic measures. So, when the subject of treatment presents itself as the logical conclusion of an examination, the physician should ask himself which of these measures, alone or in combination, is indicated for the patient before him. The items of treatment may now be briefly discussed in the order enumerated, under the caption of cardiac therapy.

Cardiac Therapy.

1. *Rest*.—The value of absolute rest as a prime therapeutic measure has been mentioned under the treatment of endocarditis, myocarditis, pericarditis, etc., hence need not be repeated here. It is well, however, to briefly draw attention to the physiology of rest. In the first place, rest lessens the demands upon the heart; it requires far less effort on the part of that organ to maintain an adequate circulation when the body is supine than when it is erect. As effort is lessened, the heart-rate is reduced. Simple changing of position from the erect to the supine posture may cause a physiologic reduction in the rate of an apparently normal heart, averaging perhaps ten beats a minute. Hence, rest in bed lessens the effort of the ventricular contraction and reduces the rate of the heart. It also lengthens diastole, the period of heart rest; it is during diastole that the coronary arteries pour their supply of nourishment into the tissues of the heart. Hence, the simple expedient of rest in the recumbent posture, by increasing the length of diastole, increases the nourishment of the heart.

The benefit of such rest is also manifest in the general circulatory tone of the patient. When a per-

son is in the erect posture gravity has a tendency to displace the level of the blood column in the vena cava. Physiologically, this level must be maintained at the highest point of the tricuspid orifice; when it falls below this point, as in the erect posture, the burden of its maintenance is thrown upon the contractile power of the veins and capillaries, supported by the muscular resistance of the abdominal wall. Rest in the recumbent posture relieves the burden otherwise imposed upon a damaged circulatory system, and an improvement in general circulatory tone results.

Mental rest has been mentioned under chronic myocardial change but emphasis might here be laid upon the actual benefit which accrues to the patient who has the reassurances of his medical attendant. The optimism, as well as the skill and common sense, of the physician has much to do with the success of treatment. In advising a patient as to his method of living and habits of life, the circumstances of the individual should be studied. It would be a lack of thought to advise a person of straitened circumstances to take an ocean voyage to secure physical rest or to avoid the cares of his occupation. It is often unwise to advise a patient who has earned his living by indoor employment to seek some occupation which exposes him to the weather, and it is as frequently equally ill-advised for the outdoor worker to secure an indoor occupation. More harm than benefit may be gained from completely revolutionizing the environment of a patient; and the mental anxiety which is brought on by attempting to earn a livelihood in an occupation or trade to which the patient is not accustomed, may far offset the beneficial results which can otherwise be

expected, and only add to the distress and unhappiness of the patient.

2. *Exercise*.—In the treatment of heart affections, exercise will be of use during the period of transition from rest in bed to accustomed activities. After the acute symptoms have subsided, the patient is usually propped in bed at short intervals during the day; when neither dizziness, fatigue nor increased heart rate follow this change of posture he may on succeeding days, be permitted to sit in a chair. Only by the adoption of such gradual change from the recumbent to the erect posture can the integrity of the circulatory balance be assured. Exercise may then be gradually permitted, and should be limited to such simple excursions as walking about the room or the house. The patient who has been in bed with an acute infection for a period of several days and who suddenly leaves his bed to go about his usual employment, runs the risk of disturbing his circulatory balance, no matter whether the cardiovascular system was affected or not during his illness.

Graded Exercise.—The application of graded exercises can be illustrated in a patient who is being treated for neuro-circulatory asthenia. After a short period of rest in bed in order that heart rate, respiratory rate and nerve balance may have an opportunity to re-adjust themselves to what is the usual range for the individual, graduated exercises may be begun. At first these should consist of a simple series of arm and leg movements, under the supervision of the physician or the nurse, and be carried up to but not beyond the point of early fatigue, as shown by increased heart response and respiratory rate. The

exercise should be gradually increased at each day's treatment until it eventuates in drills, extended walks and a degree of physical endurance that spells reconstruction for the individual.

1 Graded exercises are the keynote in the treatment of heart disease by Oertel's method, which was at one time much in vogue. This plan of treatment consisted in short excursions when the weather permitted, at first on the level, the distance to be gradually increased each day. As the endurance of the patient and as the tone of the heart improved, attempts at hill climbing were introduced as part of the treatment.

Resistance exercises have enjoyed much popularity in the treatment of chronic heart affections. This method, which is championed by Schott of Bad-Nauheim, consists in having a trained gymnastic instructor impose resistance to the action of certain groups of the patient's muscles. The patient is later able to carry on the treatment without the aid of the instructor by resisting the action of one group of muscles with the opposition of another group.¹ In connection with both the Oertel and the Schott treatment, the diet of the patient is carefully regulated; certain hours of rest are insisted upon; and elimination is stimulated by the employment of baths and by the drinking of "medicated" waters.

3. *Diet.*—The diet of a patient who is in bed with an *acute* infection of the heart shows no modification from the diet which is employed in patients who are

¹ Details can be secured from "The Gymnastic Treatment of Heart Disease," by Schott, published by P. Blakiston's Son & Co., Philadelphia.

otherwise ill with acute infections. Liquid diet is the rule in the treatment of such patients; raw eggs, broths and milk being the chief constituents.

In the treatment of *chronic* heart affections the general rule is to avoid foods in quantity and to avoid foods which are rich or indigestible, for the reason that by deranging digestion they increase the burden of the heart. When a patient's circulation is disturbed the disturbance is reflected in the gastric juices which are likely to be reduced in quality and quantity. It should be remembered, too, that gastric derangements, acting through the vagus nerve, which sends branches to both heart and stomach, may disturb the action of the heart. Again, it is possible that distension of the stomach with gas from undigested or fermenting foods may exert pressure upon the heart, although as a matter of clinical observation it is only in the exceptional instance that the area of stomach distension can be outlined by percussion. It is more probable that the disturbances commonly due to the "pressure of gas" are induced by reflex action upon the vagus nerve.

At times it is desirable to regulate the diet of patients who suffer from chronic heart affections, especially for the first few days that they are under treatment. The standard diet mentioned below is an excellent one and much in favor.

BALFOUR'S RULES FOR DIETING FOR WEAK HEARTS.

1. There must never be less than five-hour intervals between meals.
2. No solid food is ever to be taken between meals.
3. All those with weak hearts should have their principal meal in the middle of the day.
4. All those with weak hearts should have their food as dry as possible.

BALFOUR'S DIET FOR THE SENILE HEART.

Breakfast, 8.30 A.M.—Dry toast, one small piece—one or one and a half ounces—with butter; one soft boiled egg, a small piece of whitefish; three to five ounces of tea or coffee with cream and sugar, or an infusion of cocoa nibs, or milk and hot water, or cream and Seltzer. Sometimes oatmeal porridge is permissible, but not over three or four ounces should be taken.

Principal meal, 1.30 or 2 P.M.—Fish (such as haddock or sole), or meat and pudding. Two courses only are allowed. No soups, pickles, pastry or cheese. Whitefish and short-fibred meat only are allowable. The fish may be boiled in milk. A little spinach or one potato may sometimes be eaten, or a half pound of fruit, such as pears, apples, or grapes. Four to five ounces of hot water may be drunk with each meal, but no more.

5 to 6 P.M.—Three to four ounces of tea (one teacupful) infused for four minutes may be drunk, but absolutely no solid food is to be taken with it. If desirable, teaspoonful of extract of meat may be stirred in with the tea.

Supper, 7 P.M.—Whitefish and a potato or toast and pudding, or milk pudding, or bread and milk.

Bedtime.—Four to five ounces of very hot water, sipped, helps the patient to fall asleep.

4. *Massage*.—Massage is often indicated in patients who are convalescent from acute systemic infections or convalescent from acute affections of the heart itself. In chronic affections of the heart and of the arteries, as when a patient is in bed from exhaustion of cardiac reserve force massage is also beneficial. It is not advisable to manipulate the body during acute conditions, nor is it as a rule advisable to massage the thorax or the abdomen of patients suffering from cardiovascular conditions.

There are two kinds of motion, namely, active and passive. Active motion originates with the patient either as a result of his own volition or in resisting the efforts of another, as in resistance exercises. Passive motion is not under the control of the patient; it is under the control of another. Passive is the form which is most used in debilitated persons.

The varieties of passive motion include rubbing, kneading, tapping, rocking and stroking. Stroking is the procedure of choice in cardiovascular patients.

Effleurage is a term which defines a stroking movement. It causes an increase in the flow of blood to the muscles and to other soft parts, thereby increasing the circulation and facilitating the removal of waste products. It also encourages the absorption of transudates and exudates and to some degree directly stimulates the sympathetic nervous system. Effleurage stimulates the superficial muscles, produces a dilatation of the superficial vessels, stimulates elimination to a slight degree by causing an insensible perspiration, excites the skin reflexes and through the cutaneous nerves, increases rapidity of the circulation and the heart-beat in some persons. If a part be edematous and be massaged in the proper direction, *i.e.*, from the extremity toward the heart, the absorption of the fluid is encouraged by such manipulation, the size of the swollen part decreased and its muscle power increased.

Massage should not be employed beyond the powers of resistance of the patient. While to a certain point it is beneficial and stimulating it may be practised to a degree where it causes fatigue, markedly increased respirations and a decided increase in heart rate. Massage in chronic cardiovascular conditions increases the general tone of the whole body and the heart shares in the improvement. It is also true that as a result of increased tone of the heart muscle, blood passes more easily through the lungs and the respirations become deeper and easier. But it should be borne in mind that massage is simply part

of the general management of a patient who is not acutely ill; that it is but a substitute for exercise; and that it should never be employed so vigorously that it constitutes gymnastics for the patient who is in bed.

5. *Sanatorium Treatment*.—The benefits to be received from sanatorium treatment are set forth on page 207, and it is therefore not necessary to dwell upon the subject here. In passing, however, mention might be made of a therapeutic measure which is of no small importance in the modern sanatorium, namely, the cheerful atmosphere, the diversions and the social intercourse at such an institution. Patients who are morbid or unduly sensitive, or given to unwarranted fears concerning their physical condition, will find that the atmosphere of the better sanatoria is calculated to create a brighter and more hopeful mental attitude, which plays no small part in the beneficial results of sanatorium treatment.

Concerning treatment at German spas and similar resorts, it is not at all probable that such institutions can lay claim to any other benefits than those which are found in any well conducted, properly regulated institution, nor would it seem necessary for patients on this side of the water to journey across the seas for treatment which can be as well secured at home.

6. *Operative Procedures*.—Cardiovascular operations embrace the following measures: (A) paracentesis pericardii for the evacuation of serous or purulent pericardial effusions; (B) the Corradi method of wiring for the relief of symptoms, and with the hope of preventing early rupture, in thoracic aneurisms; (C) venesection, when indicated in congestion or

rupture of the cerebral vessels; in patients with dangerously high systolic pressure; and in urgent cases of auricular fibrillation, where to wait for the action of drugs is to invite death; (*D*) for the removal of foci of suppuration, which may exist in diseased tonsils or at the apices of teeth, as detailed on page 224. Other foci of suppuration, which are much less frequently the cause of cardiovascular disturbances and which may require surgical intervention, are found in discharging ears, sinus infections, gall bladder involvement, perirenal abscess, pyelitis and chronic appendicitis.

7. *Balneotherapy*.—The treatment of chronic heart affections by the employment of medicated baths has some ardent advocates. The baths are for the most part of benefit to patients of lowered vasomotor tone. It is said that the natural waters at Nauheim contain carbonic acid and that this substance slows the pulse rate and regulates the circulation of a person immersed therein, by dilating the capillaries and by indirectly stimulating the nerve centers. Under such treatment it is claimed that local congestions are dissipated, inflammatory deposits absorbed and the metabolism of the patient improved. At Nauheim the natural spring water which is heated to a temperature of from 93° to 95° Fahrenheit, contains carbonic acid, sodium chloride and calcium chloride. The treatment consists in baths which last from five to ten minutes and are usually given daily extending over a period of time varying from three to six weeks. The baths are contraindicated in acute conditions, in markedly weak or anemic patients, in those who chill easily and in those who fail to promptly react from

the initial chill upon being immersed. Thorne¹ advocates their use in angina pectoris.

It is not necessary for patients who desire such baths to journey to Germany in order to procure them. They are now available in America at certain private establishments which are under the management of competent physicians.

Schott² of Bad-Nauheim gives the following directions for the preparation of the baths at the home of the patient.

"It is now possible for those suffering from heart disease, who are not in a position to take the cure by means of the natural baths, to imitate these baths at home, to a certain extent. One should employ for this purpose preferably the natural Nauheim bath salts, or, if these are not available at the moment, make use of the most important of their saline constituents, namely, sodium chloride and calcium chloride, in the correct proportions—2 per cent. of the former, and 1 part per 1000 of the latter. These quantities may be increased when stronger baths are indicated. The carbonic acid is best obtained from sodium bicarbonate and hydrochloric acid; the chemical equivalents indicate in what proportion these ingredients are to be added to the bath. With the strong solution of hydrochloric acid (equivalent to 42.8 per cent.) equal quantities of hydrochloric acid and sodium bicarbonate should be employed. With the dilute hydrochloric acid a correspondingly larger quantity of this solution is necessary. The sodium bicarbonate, commencing with 3 ounces, and gradually increasing to

¹ Thorne: Practitioner, Aug., 1917.


² Schott: "Treatment of Chronic Heart Diseases"; Blakiston, 1914.

15, 30, or even to 45 ounces, as the baths progress, should be dissolved in the bath-water simultaneously with the other salts (sodium chloride and calcium chloride), which must also be increased in proper proportions for these stronger baths. An excess of bicarbonate of sodium is always advisable for the protection of the bathtub. After the temperature of the water has been properly regulated, an amount of hydrochloric acid equivalent to the quantity of sodium bicarbonate already dissolved in the bath is poured directly on the surface of the water from a small-mouthed bottle and distributed well over it. One should avoid any additional agitation of the bath-water, as otherwise the carbon dioxide will readily escape into the air. The layer of carbonic acid gas which forms on the surface of the water during its preparation should be driven off with a towel before the bath is used, so that the patient will not breathe it. In this way the carbonic acid gas will continue to be evolved for a considerable time, probably a half-hour or more.

"It is self-evident that even in the employment of artificial baths a constant supervision by the physician is essential, if satisfactory results are to be obtained. And often a good result is only to be secured when it is practicable to remove the patient from business and family worries into pure air and new surroundings. Suitable nourishment also plays an important part in these cases. If properly used, favorable results can be obtained with artificial Nauheim baths in a certain proportion of cases. Naturally, the number of cases to be benefited must necessarily be limited by the circumstance that the strong-

est of these baths—the effervescing and the effervescing flowing baths—cannot be imitated artificially.”

8. *Cardiac Drugs*.—The use of cardiac drugs is a therapeutic measure considered at length in the following chapter.



CHAPTER XXIV.

Cardiac Drugs.

THE drugs which have been used in the treatment of cardiovascular affections are legion; those of proven efficiency are few. Remedies which affect the circulatory apparatus in an indirect manner by their effect upon other organs cannot properly be classified as cardio-circulatory drugs; yet many such are herein considered, for the reason that they are of definite value to the practitioner in his daily work.

It is the purpose of this chapter to discuss those remedies which have been determined by laboratory research, by graphic records and by extensive clinical study to be drugs which have a definite cardiac effect. There are other preparations which have won their place in the affection of physicians through years of faithful service, which may not be mentioned in the following pages. This does not necessarily mean that such drugs are worthless; yet if they be of doubtful utility in the treatment of cardiac affections, it is better to urge the employment of drugs which have been definitely studied, as illustrated by digitalis and epinephrin. Still other drugs, that seem to be absolutely without cardio-circulatory effect, will be mentioned, not for the purpose of decrying a remedy in which many practitioners may have confidence, but more for the purpose that the physician may not in an emergency lean upon a broken reed—as I believe that doctor does who administers camphorated oil in a cardio-circulatory crisis.

DIGITALIS.

This sovereign heart remedy was first brought to the attention of the profession by Withering who wrote "An Account of the Foxglove" in 1785.¹ It is interesting to note the astute observations of this pioneer, who, in writing of the diuretic action of the drug, averred that "Digitalis seldom succeeds in men of great natural strength, of tense fiber, of warm skin, of florid complexion, or in those with a tight and cordy pulse. If the belly in ascites be tense, hard, and circumscribed, or the limbs in anasarca solid and resisting, we have but little hope. On the contrary, if the pulse be feeble and intermitting, the countenance pale, the lips livid, the skin cold, the swollen belly soft and fluctuating, or the anasarcaous limbs readily pitting upon pressure of the finger, we may expect the diuretic effects to follow in a kindly manner." With what prophetic foresight are thus confirmed the observations of today, when he allowed but little value to the drug in arteriosclerotics with high arterial pressure and associated dropsy, but admitted a beneficial action in the clinical picture of auricular fibrillation which he has so well painted! Controversial storms must have waged around Withering's head, for in closing his preface he seeks the solace of saying: "After all, in spite of opinion, prejudice, or error, time will fix the real value upon the discovery, and determine whether I have imposed upon myself and others, or contributed to the benefit of science and mankind."

¹ Withering, Wm.: "An Account of the Foxglove and Some of Its Medical Uses, with Practical Remarks on Dropsy and Other Diseases," Birmingham, 1785.

Digitalis is derived from the dried leaves of the perennial *Digitalis purpurea*, or Foxglove, collected from plants of second-year growth as they are about to flower. While many glucosides have been separated by chemists, the drug does not have an "active principle" that is universally recognized. The true therapeutic effect of the remedy is best secured by the employment of a physiologically tested tincture which combines all of the qualities claimed for several "isolated principles." The employment of "digitalis derivatives" may account for the absence of digitalis results occasionally complained of by physicians. This statement is not to be construed as expressing an unfavorable opinion of standardized tinctures or extracts as prepared by reputable pharmaceutical houses; it is intended to convey the conviction that alleged "digitalis active principles" do not give satisfying digitalis results, any more than do the stale tinctures occasionally dispensed. It should be the custom of physicians who see many heart cases to select a physiologically tested and standardized tincture of digitalis, as prepared by any one reputable pharmacist, and at the same time to assure himself of the potency of the hypodermic tablet which he proposes to use; by employing these two preparations to the exclusion of others, he soon becomes familiar with the results to be expected from a given dose, and becomes adept in the skillful employment of his remedy.

When employed in hearts of disturbed mechanism, the physiologic effect of digitalis in therapeutic dosage is shown in (1) decreased atrioventricular conduction; (2) increased force of the ventricular contraction. By decreasing the conductivity of the atrio-

ventricular node, it slows the pulse and increases the length of diastole; by increasing the force of the ventricular contraction, it increases the pulse force and raises arterial pressure. The writer is fully aware of the mass of literature relating to the action of the drug on the pneumogastric nerve, on the sympathetic fibers, and on the arterioles, yet feels that emphasis of the physiologic action, already briefly stated, is quite sufficient for the purposes of the clinician who would administer the drug understandingly.

In the form of the infusion, digitalis is prized as a remedy to relieve dropsical swellings. This it does by removing the congestion of the kidneys and by improving the blood-supply to these organs, rather than by any action on the renal cells. By thus improving the circulation of the kidneys, it is a depletant of accumulated body effusions. The infusion should be prepared as stated on page 209.

No definite limit can be placed on the amount of digitalis necessary to produce and maintain a desired physiologic result. The old rule of "giving the drug until the pulse becomes regular" has probably been responsible for many deaths by inducing heart-block, and would seem to explain the "cumulative action" and "digitalis deaths" of medical literature. When we recall that the investigations of Cohn¹ demonstrated that in some instances digitalis affected the heart within thirty-six hours of its administration, and that the effect persisted as long as twenty-two days after withdrawal of the drug (see Fig. 83), we can appreciate how unreasoning abuse of the remedy

¹ Cohn, A. E.: *Jour. Exper. Med.*, xxi, No. 6.

would induce not only heart-block, but complete exhaustion of the laboring ventricle as well.

Fairly dependable symptoms of physiologic tolerance are nausea, vomiting (which may be preceded by a sudden loss of appetite), headache and perhaps diarrhea, but unfortunately these symptoms are not

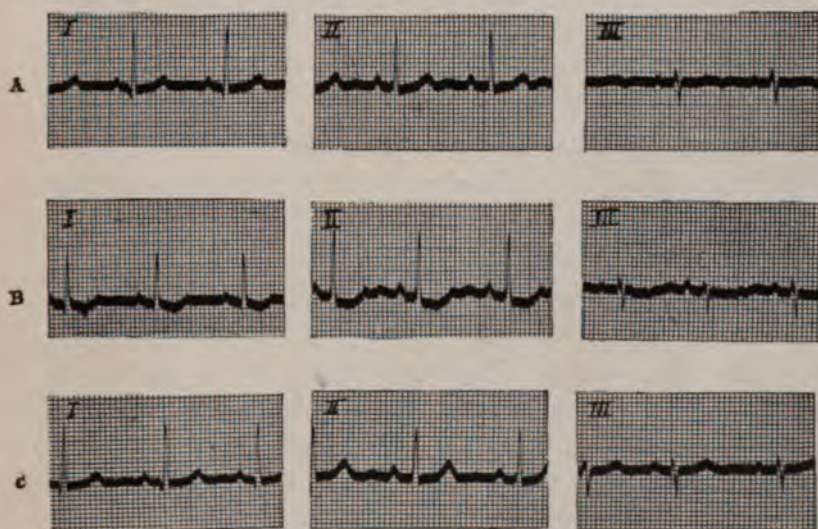


FIG. 83.—INFLUENCE OF DIGITALIS ON THE ELECTROCARDIOGRAM.

The leads in these figures are arranged horizontally, the reverse of the usual order, for ease of comparison. (A) Control curve. Before administering digitalis. (B) After 1.4 Gm. of digitalis had been given. Note (a) diminution in height of T wave; (b) downward slope from end of R or S to T. (C) This curve, taken nineteen days after the drug was discontinued, is one of a series which shows a gradual return to normal, and is the first of said series to virtually reproduce the control curve A. (Courtesy of Dr. Alfred E. Cohn, of the Rockefeller Institute for Medical Research.)

reliable guides, as they may ensue after the first dose, may not set in for several days, or may utterly fail to appear. With ordinary physical signs to guide him, the physician should revise the initial dosage of digitalis (1) upon the appearance of a gradual equality

in the ventricular and radial rates in cases of auricular fibrillation—in other words, when the pulse deficit is only ten points less than the simultaneously counted rate at the apex—administration of the drug should be carefully guarded; (2) upon the appearance of “digitalis coupling,” often revealed upon thoughtful radial study, or on auscultation, in patients in whom the drug is used.

It is almost trite to remark here that prolonged use of digitalis over periods of months is to be condemned, unless frequent professional observations so dictate. One patient recently seen had been taking digitalis upon his own initiative, for a year and a half. A *non-repetatur* upon a prescription will save a physician from the censure attached to the medical attendant who wrote the prescription in this instance.

Digitalis is the remedy par excellence in auricular fibrillation, in which condition its chief beneficial effects are observed, and upon which its reputation rests. It is of marked value in auricular flutter. Paroxysmal tachycardia, when it induces exhaustion of the cardiac muscle, may call for the support afforded by this drug, as also may pulsus alternans. Heart-block is a contraindication to digitalis, unless the block be complete, in which latter event it may increase cardiac tone. The only contraindication to digitalis administration in *complete* heart-block is the presence of the Stokes-Adams syndrome. Cardiac enlargement does not call for its employment unless circulatory failure ensues.

If the physician will but think of digitalis as a drug to be used only when especially indicated for definitely recognized conditions, both laity and pro-

fession will be benefited by the thought. The detection of murmurs does not call for digitalis, although the drug may, of course, be indicated in valvular lesions with evidence of exhaustion of the cardiac muscle.

Dosage.—It is customary to give digitalis in a much larger dose than was formerly the rule. It is not unusual, in a cardiac emergency to see it administered in 30 minim* doses at three hour intervals until perhaps as much as 3 drachms have been taken in the course of 24 hours. When the drug is given in such a large dosage it is necessary that the patient be carefully watched and that the administration be discontinued upon the appearance of any of the symptoms of physiologic intolerance which are mentioned above, concerning which Withering says, "Let the medicine be continued until it either acts on the kidneys, the stomach, the pulse, or the bowels; let it be stopped upon the first appearance of any one of these effects."

STROPHANTHUS.

This drug of the digitalis group is derived from the seeds of the African plant *Strophanthus hispidus* or *kombé*. *Strophanthus* is believed to be similar to digitalis in its effect on the heart. It is often administered to patients who have an idiosyncrasy to digitalis or in those in whom digitalis produces early gastrointestinal symptoms—yet it is not always without this untoward effect itself. If tincture of *strophanthus* is given to a patient who has previously had courses of digitalis it should be very cautiously em-

* Pratt reminds us that it takes from 35 to 40 drops from an ordinary medicine dropper to equal *fifteen minims* (which equal 1 c.c.).

ployed, as it is likely to *produce sudden death* under such circumstances.

An explanation of this treacherous action of the tincture of strophanthus may be found in the relatively small size of its minimum lethal dose for animals and in the *variable absorbability* of the drug when given to man by the mouth. The United States Pharmacopœia, in its 9th edition, for some strange reason requires that strophanthus shall be one hundred times as active as digitalis. Both are physiologically tested by the same method; the average dose of each tincture is given as 8 minims: and yet strophanthus is to be so prepared that it will arrest the heart of a frog, in systole, in a dose which is 100 times smaller—hence a hundred times stronger—than is required of digitalis!

As to absorbability, there is no clinical method of determining beforehand how much, if any, of a drug given by mouth will be absorbed or *when* it will be absorbed. Therefore, if tincture of strophanthus be made so potent on the theory that only a *fraction* of the drug will be absorbed, the premise is certainly false and the conclusion impractical, as witnessed by the unexpected deaths that may suddenly follow the administration of tincture of strophanthus.

With such a discrepancy existing in the preparation of tinctures that have a similar effect upon the heart, the physician should take pains to assure himself that he is not giving a patient a dose of strophanthus which is one hundred times more active than the dose of digitalis which he has previously been using. Indeed he would do well to avoid the use of tincture of strophanthus altogether, especially if the patient has

already had a course of digitalis medication, for fear of sudden and unexpected death of the patient.

The active principle of *strophanthus*, which is *strophanthin* (amorphous) is used intravenously, in a strength varying from $\frac{1}{500}$ to $\frac{1}{125}$ grain, the last mentioned dose being indicated only in urgent cases. Improvement in cases of desperate circulatory embarrassment may be expected to follow the intravenous injection of *strophanthus* within a few moments. The drug should not be repeated within 24 hours, nor should it be routinely employed intravenously.

EPINEPHRIN.

The active constituent of the suprarenal glands was first isolated by Abel who gave it the name of epinephrin. The substance, while elaborated by ductless glands, enters the systemic circulation in all vertebrates by the way of the suprarenal blood-vessels. Various names have been given to the suprarenal extract, the terminology depending upon the choice of the manufacturing chemists who issue it, such as adrenalin, suprarenalin, epinin, etc. It is a dependable but fleeting remedy in circulatory collapse.

The action of the suprarenal substance upon the heart is shown by the experiments of Stewart and Rogolf,¹ who interfered with the entrance of the suprarenal blood into the circulation, and thus increased the irregularity of a heart. Upon readmitting the suprarenal blood stream to the circulation it was found that the irregularity was corrected. They further demonstrated, by artificial administration of

¹ Stewart and Rogolf: Jour. Pharmacology and Experimental Therapeutics, July, 1919, xiii, 4.

epinephrin, that it is the active constituent in the suprarenal blood.

Epinephrin is indicated where there is evidence of acute circulatory collapse with falling blood-pressure. The marked rise in blood-pressure which it induces is due to direct action on the muscle of the vessel walls. While all of the arteries are constricted by epinephrin, the greatest constriction is found in the vessels of the splanchnic area. The marked accentuation of the heart is due to stimulation of the accelerator nerves which are distributed in the heart muscle.

Epinephrin is fleeting in its action, and the rise in blood-pressure which is observed when the substance is administered, generally falls to normal or less than normal within five minutes after a moderate dose is given. For this reason the drug should not be used where a sustained circulatory result is desired. Janeway used the large dose of one drachm of a 1 to 1000 solution with most amazing results in the restoration of a patient apparently moribund.

The physiologic action of epinephrin as outlined above, follows the *intravenous* use of the substance. Such effects will not be observed when the cruder preparations of the drug, such as the desiccated glandular substance, is administered by the mouth.

THE NITRITES.

Nitroglycerin, amyl nitrite, and sodium nitrite are dependable circulatory aids where immediate effect is desired; their action is, however, fleeting in character, the reaction not being long sustained, and for this reason they are administered at two or three hour intervals.

The nitrites cause acceleration of the pulse by a reflex action on the vagus center in the medulla. There is no experimental proof that they have any direct action upon the heart-muscle; hence a damaged myocardium does not contraindicate their employment when their action is otherwise desired, as in relieving the spasm of angina pectoris. In this condition the dose of nitroglycerin can be larger than customary, as mentioned on page 348. We should think of the nitrites as relieving arterial spasm, for it is in this direction that they exert their most pronounced therapeutic effect. The value of this form of medication in the cardiovascular failure of acute infections, such as lobar pneumonia, is to be also borne in mind.

The dose of the nitrites is as follows: The spiritus glycerylis nitratis is a 1 per cent. aqueous solution of nitroglycerin, and is given in 1- or 2-minim doses, sometimes gradually increased. Tablets of nitroglycerin (*tabellæ trinitrini*) each contain $\frac{1}{100}$ grain. The *perles* of amyl nitrite contain 5 minims each, and are to be crushed in a handkerchief and inhaled for the relief of arterial spasm.

ATROPINE.

This alkaloid of belladonna is extracted from the roots or leaves of "deadly nightshade." Though at first slowing the heart by stimulation of the vagus center in the medulla, this slight initial effect soon passes off, and the heart-rate increases, owing to a paralysis of the terminal inhibitory fibers of the vagus nerve. Atropine probably increases the conductivity of the atrioventricular bundle; hence it is the remedy indicated in all degrees of heart-block, the subcutane-

ous dose of the sulphate being $\frac{1}{50}$ grain, to be repeated when the effect has disappeared. The action of the drug on the bundle to some degree neutralizes the effect produced by giving digitalis in excess.

Tincture of belladonna is administered in the dose of 5 to 40 minims. The drug produces dryness of the fauces, dilated pupils, sometimes an erythematous rash, and perhaps a talkative delirium, in full medicinal dose.

MORPHINE.

This drug slows the heart-rate, not by a direct action upon the cardiac muscle—for there it has no effect—but by stimulation of the vagal center. Hence, myocardial lesions do not contraindicate the drug, except in so far as its effects upon other parts of the body add to the heart's embarrassment; morphine checks all bodily secretion except that of the skin.

The pain of myocarditis and angina pectoris, the rate in tachycardias when enhanced by excitement and restlessness, the intractable insomnia sometimes present in heart affections and the dyspnea of others, all may call for the employment of morphine.

Morphine sulphate is administered in doses of $\frac{1}{8}$ to $\frac{1}{4}$ grain, repeated at two- or three-hour intervals, until the desired result is secured. When indicated at all, morphine is *indicated to effect*, a point discussed on page 239.

ANTISYPHILITICS.

To include such drugs as salvarsan and neosalvarsan—for which the Federal Trade Commission has adopted the names “arsphenamine” and “neoarsphenamine” respectively,—under the heading of

cardiac drugs is perhaps an unusual procedure, but one which is nevertheless warranted, when one considers the frequent etiologic relation between syphilis and cardiovascular disease. When there is need for the remedies it would be well to consult the laboratory or a pharmaceutical chemist for the more recent methods of preparing and administering the drugs. Refinement in preparation and improvement in the technique of administration are being constantly evolved, and any method stated in these pages would soon be supplanted by more recent and more approved methods.

THE IODIDES.

The iodides, the most familiar preparation of which is the iodide of potassium, are valuable adjuncts in the treatment of circulatory disorders, not only by virtue of their action in specific disease, but also as the result of their alterative effect. Under Arteriosclerosis (page 305), administration and dose are discussed.

STRYCHNINE.

The modern school of drug investigation takes issue with the clinical experience of countless physicians as concerns the action of strychnine upon the circulation. Thus Neuburg¹ in his experiments, found that patients were not benefited by strychnine; the symptoms of circulatory failure were not improved in the slightest: and this observer concluded that neither the pharmacologic nor clinical evidence justifies the use of the drug in the treatment of acute or chronic heart disease. Pilcher and Sollman² demonstrated

¹ Neuburg: *Amer. Jour. Med. Sci.*, cxlix, 696.

² Pilcher and Sollman: *Jour. Pharmacology and Experimental Therapeutics*, vi, 331.

that strychnine was without effect upon the heart; it had no direct action upon the blood-vessels; and that it produced no marked effect upon blood-pressure.

Despite these and many other investigations in which strychnine is found to be without demonstrable effect, the fact remains that it has for generations been successfully used by physicians in the treatment of cardiovascular conditions. These dissenting views can be reconciled when it is understood that strychnine actively stimulates the secretion of the suprarenal glands, and while the effect on the suprarenal extract is fleeting, the internal secretion nevertheless causes a rise in blood-pressure and markedly accents the action of the heart, due to the stimulation of the accelerator nerves. If there were circumstances which interfered with the elaboration of suprarenal secretion, such as exhaustion or disease of the suprarenal glands, one would not expect strychnine to be of any benefit in stimulating the circulation of a patient so affected.

Strychnine does have a stimulating effect upon the suprarenal gland as pointed out by Sajous¹ in 1903 who stated that "strychnine, of all drugs, stands prominently as the most active suprarenal stimulant." Stewart and Rogolf² were able to demonstrate that strychnine caused a marked output of epinephrin from the suprarenal glands, ten times the original amount having been observed in some instances. Doses of strychnine well within the therapeutic range, and which cause little or no exaggeration of reflex excitability, are capable of producing a considerable augmentation in the rate of output.

¹ Sajous, C. E. de M.: *Internal Secretions*, i, 43.

² Stewart and Rogolf: *Jour. Pharmacol. and Exper. Therap.*, xiii, May, 1919, No. 2, pp. 95-166.

Inasmuch as there might be affections of the ductless glands in which strychnine could not be depended upon to stimulate suprarenal secretion, reliance should not be placed in the drug as an *emergency* remedy. In cardiac collapse it would be better therapeutics to employ epinephrin intravenously. In patients suffering from a loss of vasomotor tone or from myocardial exhaustion, strychnine has always been a valued remedy. It also finds splendid employment during the period of convalescence, especially when combined with iron and quinine in familiar proportions. Sulphate of strychnine is administered in the dose of $\frac{1}{40}$ to $\frac{1}{20}$ of a grain 3 times daily after meals.

CAFFEINE.

As a result of their animal experimentations, Pilcher and Sollman¹ conclude that caffeine causes: (a) cardiac stimulation; (b) increase of heart-rate not due to vagus depression; (c) vasodilatation through peripheral depression of the vasoconstrictor mechanism; (d) central vasoconstrictor stimulation to be generally ineffectual. From this can be deduced the tenable hypothesis that caffeine probably accelerates the heart action by direct stimulation of the heart-muscle. Caffeine is administered in dosage of from 2 to 4 grains.

ALCOHOL.

Alcohol raises for a few moments the systolic pressure, and thus acts as an apparent circulatory stimulant; it cannot, however, be regarded as a true circulatory stimulant, inasmuch as it decreases cardiac effi-

¹ Pilcher and Sollman: Jour. Pharmacol. and Exper. Therap., iii, 89.

ciency, raises disproportionately the diastolic pressure, and lowers pulse-pressure, according to the exhaustive investigations of Lieb,¹ corroborated by many others. Alcohol is no longer considered a food, for it has been determined that its oxidation in the body is a protective oxidation (as is that of uric acid, xanthin bodies, leucin, etc.); it is not oxidized for the purpose of being used by, or stored up in, the economy as a food.²

CAMPHORATED OIL.

There is no conclusive evidence that camphorated oil stimulates heart-muscle. There is no evidence that it raises the blood-pressure. Nor do clinical studies indicate that it materially alters the pulse-rate or the force of the ventricular contraction. With patients in whom the hypodermic administration of 5 grains of camphor in sterilized almond oil was utterly unattended by a definite therapeutic result, the administration of a dependable tincture of digitalis in sufficient dosage has been observed to increase the force of ventricular contraction and to improve general circulatory tone of the patient.

Camphorated Oil Tumors.—The subcutaneous use of camphorated oil may give rise to inflammatory tumors at the site of injection. According to Mook and Wander,³ they do not usually develop until a period of months has elapsed following the injection. The inflammatory tumor presents the characteristics of heat and redness. The mass, while tender, is not especially painful. It is linear in outline varying

¹ Lieb: Jour. Am. Med. Assn., lxiv, 898.

² Hall: Medical Times, xlv, 39.

³ Mook and Wander: Jour. Cutan. Dis.

from the size of a walnut to that of an orange and is usually lobulated. Sharp, well defined lines separate the borders of the camphorated oil tumor from surrounding tissue. Commenting on the use of camphorated oil, these two observers mention that pulmonary emboli have followed the injection of liquid petrolatum and conclude, that it is dangerous to use liquid petrolatum as a vehicle for any remedy which is to be injected in subcutaneous tissue.

ANESTHETICS IN HEART AFFECTIONS.

The physician is frequently called upon to decide whether an anesthetic should be administered to persons suffering from heart affections, and is often required to express an opinion as to the anesthetic of choice. A brief consideration of the cardiovascular action of the three general anesthetics—chloroform, ether and nitrous oxide—may be of aid in forming an opinion as to the anesthetic of choice when anesthesia is absolutely necessary.

Chloroform.—Chloroform affects the heart, the vessels, the blood-pressure and the cardiac nerves. The changes which it induces at first may not be noticeable in the pulse, should the rhythm be unaltered, and one may fail to note on palpation that the volume of the blood-wave is reduced. Chloroform frequently induces a *gradual* cardiac depression. The force of the heart's contraction is lessened by the action of the drug on the cardiac muscle and the heart relaxes in tonicity. The auricles may gradually become so depressed that their feeble contraction plays no part in driving the blood into the ventricles; it is at this stage that the pulse becomes slow. Eventually

the ventricle muscle may be overpowered and the heart arrested in diastole. Embly has noticed that the heart may also stop beating on account of vagal stimulation under chloroform, and fail to resume contractions after the removal of the anesthetic.

Cardiac syncope is another accident that may suddenly intervene when chloroform is administered. Cardiac syncope occurs with greater frequency in the earliest stages of anesthesia, and has been oftener noted where the steady administration of the anesthetic has been subject to interruption. Levy¹ believes that cardiac syncope is due to ventricular fibrillation. The advent of cardiac syncope may be preceded by premature contractions, tachycardia, or other pulse irregularities.

Chloroform lowers the blood-pressure by dilating the vessels of the splanchnic area, but principally for the reason that it weakens the cardiac contraction. It should be remembered, that chloroform depresses the heart to a degree 25 to 30 times greater than ether.

The kidneys are affected by chloroform, albuminuria having been noted by various observers in from 8 to 30 per cent. of the people upon whom it is used. Under both ether and chloroform the secretion of urine is diminished and usually remains scanty several hours afterward.

The contraindications to the employment of chloroform in cardiovascular conditions may be summed up as follows: (1) patients with evidences of myocardial degeneration; (2) persons in whom the systolic blood-pressure is unusually low; (3) coincident renal involvement; (4) chronic valvular disease—

¹ Levy: *Heart*, 1913, iv, 219.

not that the valvular lesion itself is of especial import, but for the reason that valvular lesions may be safely considered as an evidence of some degree of heart-muscle involvement; (5) atheromatous arteries, as they are liable to rupture during the stage of excitement and produce apoplexy; (6) anemia. It has been stated by DaCosta that chloroform should not be used on any patient in whom the hemoglobin is less than 50 per cent. Chloroform should not be used on a patient to whom adrenalin has been administered intravenously, as the combination may induce ventricular fibrillation, a condition which usually promptly terminates in death.

Ether.—Ether produces the same effect upon the heart and vessels as does chloroform, but to an infinitely lesser degree. Cardiac syncope is a rare event in ether anesthesia. Cushny states that if the ether inhaler is not applied too closely and asphyxiation of the patient avoided, there is very little, if any, albuminuria attendant upon ether administration.

Nitrous Oxide.—Nitrous oxide is the safest anesthetic for use in a patient suffering from heart affections. While the gas raises the blood-pressure, and may thus induce apoplexy, the possibilities of blood-pressure increase are reduced to almost *nil* if nitrous oxide be diluted with oxygen, in customary proportions.

CHAPTER XXV.

Cardiac Camouflage.

ONE who devotes much time to cardiac conditions will occasionally have his attention arrested by unconventional peculiarities in an apparently normal heart. While of course no two hearts are exactly alike, those which experience has taught the physician to classify as within normal limits may be expected to behave in a preconceived and rational manner. Likewise, a diseased heart manifests itself by certain symptoms and signs which experience has taught one to classify as distinctly cardiopathic. Between the extremes of health and disease there is a middle group of disordered heart actions, usually of brief duration and quite out of character with accepted standards, which at once arouse the suspicions of the examiner as being artificially induced. This article sets forth the manner in which some heart deceptions may be clinically recognized with a satisfying degree of certainty, without recourse to the laboratory facilities of either the cardiographist or toxicologist.

THE REASON.

Individuals in military or in civil life, on occasions, believe that they can escape from some odious circumstance confronting them by tampering with the normal action of their heart, and so secure a physician's certificate of disability. A recruit may thus attempt to evade the operations of the draft law; an unwilling witness may thus hope to avoid a summons to civil

court; women who are adrift on a sea of domestic troubles may, with the idea of bringing a recalcitrant husband to his senses, feign illness by disordering heart action with drugs. It requires no mean degree of cleverness to conceive of and to simulate cardiac disease; the deception also demands no small amount of courage in execution: consequently, such people are adroit and difficult to handle.

THE MEANS EMPLOYED.

The simplest form of such dissembling is exhibited by the person who rushes to the examining room with hand clasped over a tumultuous precordium and an assumed anxiety of countenance, panting for breath from some violent muscular exertion through which he has just put himself. "Cooling his heels" in the waiting room for half an hour while waiting for a second examination, effectually disposes of this type of nuisance. In another group belongs the soldier who, with implicit faith in the popular street notion that tobacco causes heart disease, deliberately saturates himself with the weed prior to examination. In the writer's experience, that variety of cardiac arrhythmia which may be thus induced is sinus arrhythmia, especially noted in the recumbent posture, and to which the recruit may direct the attention of the examiner. This tobacco arrhythmia disappears immediately following exercise, re-appearing as the heart-rate approaches normal. The rate response to exercise is well balanced; no murmurs are induced; the transverse diameter of the heart is within usual limits. If one will examine such a person when he has

had no opportunity to prepare himself the arrhythmia will have quite disappeared.

Acetanilid is a drug universally available in the form of headache powders. It induces, in overdose, moderately severe cyanosis, slow but regular pulse, enfeebled ventricular systoles, a suggestion of mental haze and a sluggish cardiac response to exercise. The symptoms will clear up within a few hours if the patient is carefully watched to prevent further abuse of drugs.

Atropine suggests itself in those tachycardias where the rate remains persistently over a hundred-and-twenty and where said rapid rate varies with postural change. This circumstance is quite at variance with the pulse in true paroxysmal tachycardia, where the rate remains essentially the same whether the patient be erect or recumbent, and where there is no marked alteration produced by either exercise or prolonged rest. The respiratory rate under atropine is increased by exercise; it is little changed in paroxysmal tachycardia. In drug tachycardia the heart sounds, while rapid, give the impression of being clearly struck; in tachycardias of other origin they are often so blurred as to suggest a search for additional evidence of valvular disease. In suspected atropine tachycardia one should not fail to look for the evidence from which "bella-donna" derives its name—for the flushed cheek, sparkling eye, dilated pupil, enlivened lips and animation which commend the drug to some women. Two or three days of careful watching may be required to satisfactorily clear up a drug tachycardia.

Digitalis.—Despite the curious notion that digitalis is without effect on a normal heart, the writer is confident that he has met with instances of digitalized hearts where there was no evidence of cardiac disease. In one particular instance it was impossible to secure the stoutly withheld confession of drugging, which the following clinical observations certainly suggested. The coupled pulse was typical; the rate was 64; pulse full and strong, arteries elastic. The maximum cardiac impulse, located in the 5th interspace, $7\frac{1}{2}$ cm. from the midsternal line, was slow and forceful, greeting the hand with a deliberate thrust. There was no pulse deficit. The transverse diameter of the heart was within usual limits (11.5 cm.); the heart sounds, while paired, were unaltered save for an increased muscular quality of unusual duration to the mitral first. Examination in the recumbent posture showed no change in rate, rhythm, volume, intensity or duration from the preceding notations, made with the patient erect. The coupled beats were so persistent and characteristic that opportunity was extended to 3 colleagues to study the phenomenon; nor did the pulse show any change under the varied methods of approach of these observers. The exercise test of hopping was then employed, the person exercising with an agile ease and with an avidity quite out of keeping in one who rather prided himself on the desperate condition of his heart. Immediately after exercise the coupling disappeared and the pulse assumed a normal rhythm, at a rate of 120; dyspnea was not marked. Two minutes after exercise the rate was still perfectly rhythmical at 84 beats per minute. During the succeeding eight days, while the patient

was under constant surveillance, the irregularity would recur at progressively-decreasing intervals and finally disappeared altogether.

SUSPICIOUS ATTITUDES OF MIND.

In army service and in athletics, the vast majority of young people of both sexes regard a cardiac examination as a perfunctory regulation to which they indifferently submit, confident in the self-assurance of youth that their hearts are perfectly normal. Comparing, as they do, their physical condition as yet free from symptoms of circulatory failure, with the advanced heart wrecks which they have known, there is ground for the assurance. It is also quite natural for other individuals, once attention has been drawn to their hearts, to manifest an interest in the progress and outcome of a final examination: but a too lively interest should arouse suspicion. He who complains of pain which his tranquil eye fails to reflect; he who prates of the number of times he has been rejected from beneficial societies; he who is too alert in obeying directions, instinctively puts the physician on guard to detect some form of cardiac camouflage. There are two stumbling blocks in the dissembler's fabrication of heart disease over which he will usually fall flat; one is previous history. An impostor does not seem to know that actual cardiac fault is quite constantly the result of antecedent infection or of long-continued physical or emotional strain; consequently, the important detail of sufficient provocative history is lacking in his fabrication. The second stumbling block is the attitude with which he greets the instruction to "hop 100 times on one foot"; usu-

ally there is a profoundly grave, negative shaking of the head as he proceeds martyr-like, as though in exquisite pain; or, he may determinedly spring at the welcome opportunity to demonstrate heart disease by the most exaggerated and exhausting physical effort, little realizing that damaged hearts cannot always finish the test. The final decision as announced to the secretary, "Heart within the limits of health; accept for general military service" may be greeted with an ill-concealed resentment on the dissembler's part, which is rather gratifying to the suspicions of the examiner.

GENUINE MENTAL ATMOSPHERES.

If a physician be at all susceptible to atmospheres—and most are—he cannot fail to grasp the impression of something distinctly genuine when in the presence of an actual case of heart disease. Not that the appearance of the patient need be especially suggestive; nor is the impression due to the gravity of the symptoms; it does not arise from convincing statements on the part of the sufferer; it is not always based on the especially serious import of a sign or group of signs. There is something elusive in the patience, in the forbearance, in the glance, in the resignation—in the partial absolution from temporal things—that suggests a touch of the supernatural in the actual cardiopath. It is the mental atmosphere of the heart patient that casts about him the aura of genuineness.

INDEX

	PAGE		PAGE
a WAVE	95	AIDS IN BLOOD-PRESSURE	130
absence of	100	ALCOHOL	38, 407
in radial tracing	101	ALCOHOL AND ARTERIOSCLERO-	
split a	101	SIS	282
a WAVE OF PHELEBOGRAM	95	ALTERNATION OF THE PULSE.	
ABERRANT WAVES	118	See PULSUS ALTERNANS .	57
ABNORMALITIES OF THE CAR-		ANASARCA	28
DIAC MECHANISM. See		ANATOMY OF THE HEART	4
IRREGULAR PULSE	134	ANESTHETICS IN HEART AF-	
ABDOMINAL SUPPORT		FECTIONS	407
for relaxed abdominal ves-		ANEURISM	307
sels	133	effect on circulation	316
ABSCISSÆ OF ELECTROCARDIO-		definition of	307
GRAM	112	diagnosis of	324
a-c INTERVAL	97	diet in	333
ALBUTT, CLIFFORD	290	etiology of	308
ACCELERATOR NERVES	20	general symptoms of	316
ACCENTUATIONS	76	physical signs of	322
ACETANILID, ABUSE OF	414	pre-aneurismal stage	309
ACTIVITIES, RESUMPTION OF ..	383	pressure symptoms	316
ACTUAL DROPPED BEATS <i>vs.</i>		prognosis of	325
PREMATURE CONTRAC-		rupture of	328
TIONS	158	thoracic	312
ACUTE INFECTIONS OF CHILD-		treatment of	332
HOOD	34	varieties of	307
ACUTE MYOCARDITIS		wiring of	334
diagnosis of	197	"ANEURISM OF SIGNS"	316
etiology of	189	ANEURISM OF SYMPTOMS	318
treatment of	204	ANEURISMAL "EROSION"	323
ACUTE PERICARDITIS	167	ANGINA ARTERITIS	343
ADHERENT PERICARDIUM	182	ANGINA ABDOMINALIS	342
ADHESIONS, EXTRAPERICARDIAL	179	ANGINA PECTORIS	337
ADHESIONS, INTRAPERICARDIAL	179	diagnosis of	345
ADHESIONS, PERICARDIAL,		etiology of	339
PHYSICAL SIGNS OF	180	objectionable terms for ...	338
ADOLESCENCE		pathology of	340
infections of	35	physical signs of	345
"ADOLESCENT HEART." Also		prognosis of	346
see NEURO-CIRCULATORY		symptoms of	343
ASTHENIA	38, 352	the condition defined	337
ADRENALIN	401	treatment, palliative	348
ADVENTITIA OF ARTERY	279	treatment, prophylactic ...	347
AFTERNOON REST, VALUE OF..	373	ANGINA SINE DOLORE	338
		ANGOR, IN ANGINA PECTORIS.	338
		ANTISYPHILITICS	404

	PAGE		PAGE
AORTA		ARTERIAL THICKENING, DE-	
dilatation of	311	GREES OF	294
percussion of arch	60	ARTERIES	
proximity to spine	263	adventitia of	279
AORTIC INSUFFICIENCY	254	artificial production of	
arterial sounds in	260	sound	76
auscultation in	259	beaded	294
blood-pressure in	261	calcified	294
capillary pulse in	257	coronary	20
diastolic murmur in	259	distributing	280
essential signs in	261	"goose neck"	294
etiology of	256	infiltrated	294
pallor of	257	intima of	279
palpation in	258	locomotor	294
percussion in	258	media of	279
physical signs of	256	"pipe stem"	294
sudden death in	255	sounds in	76
"water-hammer" pulse	258	"silver wire"	296
AORTIC REGURGITATION. See		supply	280
AORTIC INSUFFICIENCY		thickening of	48
AORTIC STENOSIS	262	vaso-vasorum	279
diagnosis of	263	"whip cord"	294
physical signs of	263	ARTERIOGRAM	91
rarity of	262	diastolic period	95
AORTIC VALVES, PROXIMITY TO		dicrotic notch	95
BUNDLE	158	interpretation of	97
AORTITIS, ACUTE	309	normal	95
diagnosis of	312	percussion wave	95
etiology of	310	ARTERIOSCLEROSIS	278
physical signs of	311	blood-pressure in	290
symptoms of	310	circulatory effects of	279
AORTITIS, SYPHILITICA	310	clinical recognition of	286
APPARATUS		definition of	278
blood-pressure	127	drugs in	303
for breathing exercises ...	212	etiology of	281
electrocardiographic	122	eye signs in	295
orthodiascope	87	heart in	292
ophthalmoscope	84	kidneys in	291
polygraphic	90	prognosis of	296
stethoscope	71	pulse in	293
APPENDICITIS	37	treatment of	298
APHORISM, CARDIAC	3	ASCITES	28
APICAL PRESYSTOLIC MURMUR	252	ATHEROMA OF VALVES	244
ARBORIZATION BLOCK	156	ATHEROMATOUS PLAQUES	278
ARCHITECTURE OF HEART . 7, 8, 9		ATTITUDE	
ARCUS SENILIS	295	of patient	29
ARSPHENAMINE	404	of physician	30
ARTEFACTS		ATRIO-VENTRICULAR BLOCK ...	156
in electrocardiogram	122	ATRIO-VENTRICULAR BUNDLE ..	16
in polygram	97	ATRIO-VENTRICULAR NODE	15
ARTERIAL PAIN	340	ATRIO-VENTRICULAR RHYTHM .	162
ARTERIAL SOUNDS	260	ATROPINE	403
		ATROPINE, ABUSE OF	414
		ATROPINE IN HEART-BLOCK ..	160

	PAGE		PAGE
AURICLE		BELLADONNA	403
posterior	10	BIGEMINY	
systole of	15	frequent cause of	101
AURICULAR		illustrations of	139
auricular-ventricular bundle	16	<i>vs.</i> premature contractions.	101
fibrillation	17, 49	BILLINGS, FRANK	186
septum	12	BLOCK	156
systole	15	arborization	156
AURICULAR ACTIVITY IN		bundle branch	156
HEART-BLOCK	159	causes of	157
AURICULAR FIBRILLATION	145	complete	157
blood pressure in	149	definition of	156
capillary method in	149	degrees of	157
clinical recognition of	147	delayed conduction	157
definition of	145	diagnosis of	158
digitalis in	151	dropped beat, actual	157
electrocardiogram of	115	functional	156
etiology of	146	fundamental concept of	158
historical	145	high grade	157
in polygram	99	location of	156
polygram of	98	low grade	157
preceded by tachycardia	140	organic	156
prognosis of	150	prognosis of	159
summary	155	sino auricular	156
the fibrillation triad	148	Stokes-Adams syndrome	158
treatment of	151	transient	156
volume in	57	treatment of	159
with mitral stenosis	250	two to one	157
AURICULAR FLUTTER	144	varieties of	157
changes to auricular fibril-		BLOOD	
lation	145	circulation of	14
electrocardiogram of	114	BLOOD CULTURES IN CARDIAC	
in electrocardiogram	124	DIAGNOSIS	83
in polygram	101	BLOOD-PRESSURE ESTIMATES	126
symptoms suggestive of	144	blood-pressure aids	130
AURICULAR STANDSTILL	162	blood-pressure "don'ts"	131
AUSCULTATION	70	factors in	131
immediate, in aortic insuffi-		importance of comparative	
ciency	70	readings in	127
immediate, in heart-block	70	technique	129
place of	1	the apparatus	127
purpose of	70	treatment of high blood-	
technique	70	pressure in	132
AUTOGENOUS VACCINES	239	usual standards	130
AVERAGE MEASUREMENTS OF		BLOOD-PRESSURE IN AURICULAR	
HEART	5	FIBRILLATION	149
BALFOUR'S DIET	386	BLOOD-PRESSURE IN NEURO-	
BALNEOTHERAPY	389	CIRCULATORY ASTHENIA	358
BALL PLAYING	376	"BLUE BABIES"	12
BASAL SYSTOLIC MURMURS	79, 259	BIMANUAL PULSE ESTIMATES	56
BATHING	370	BOXING	376
BATHS, MEDICATED	389	BRADYCARDIA	50
BEADED ARTERIES	294	BREATHING EXERCISES IN	
		MYOCARDITIS	212

	PAGE		PAGE
BROADBENT'S SIGN	44	CARDIAC IRREGULARITIES FORE-	
BRUIT, CARDIAC	74	SHADOWING HEART FAIL-	
BRUIT DE ROGER	274	URE	163
BULB, JUGULAR	91	CARDIAC MECHANISM, ABNOR-	
BUNDE BRANCH BLOCK ...	77, 156	MALITIES OF. See Irregu-	
BUNDLE OF HIS, RATES OF ...	18	LAR PULSE	134
"BUTTON HOLE MITRAL"	244	CARDIAC NERVES	19
		accelerator	20
CACODYLATE OF SODIUM, IN		inhibitory	19
PERICARDITIS	186	plexus, cardiac	20
CAFFEINE	407	CARDIAC SLEEP START	
CALAMITIES, EFFECT OF ON		orthopnea	27
HEART	37	CARDIAC THERAPY. See	
CALCIFICATION OF ARTERIES ..	294	THERAPY, CARDIAC	381
CAMERA, OF ELECTROCARDIO-		CARDIOGRAM	91
GRAPHIC APPARATUS	107	CARDIO-RESPIRATORY MURMURS	78
CAMPBORATED OIL	408	CARDIOSCLEROSIS	196
CAMPBORATED OIL TUMORS ..	408	"CARDIOVASCULAR RENAL Dis-	
CANE SUGAR IN HEART AF-		EASE"	292
FECTIONS	211	CAUSES OF HEART-BLOCK	157
CAPACITY OF HEART STRUC-		"CEREBRAL" FORM OF ENDO-	
TURE	12	CARDITIS	233
CAPPED TEETH AND ENDO-		CHAMBERS OF THE HEART ...	7
CARDITIS	227	CHANGE OF POSTURE, EFFECT	
CAPILLARY FIBROSIS	279	OF	383
CAPILLARY METHOD IN AURIC-		CHEYNE-STOKES BREATHING .	29
ULAR FIBRILLATION	149	CHILDBIRTH	379
CAPILLARY PULSE	257	CHILDHOOD	
CARDIAC APHORISM	3	acute infections of	34, 190
CARDIAC CAMOUFLAGE	412	"growing pains" of	221
genuine mental atmosphere.	417	infections of	366
suspicious attitudes of mind.	416	pericarditis during	168
the means employed in....	413	usual incidence of acute in-	
the motive for	412	fections in	192
CARDIAC CYCLE	18	CHILD, MANAGEMENT OF, IN	
CARDIAC DRUGS	393	HEART AFFECTIONS	367
alcohol	407	CHLORIDE BATHS	389
anesthetics in heart affec-		CHLOROFORM	409
tions	409	CHOREA	34, 35
antisyphilitics	404	CHOREA AND ENDOCARDITIS ...	222
atropine	403	CHRISTIAN HENRY A.	171
caffeine	407	CHRONIC MYOCARDIAL AFFEC-	
camphorated oil	408	TIONS	193
digitalis	394	CHRONIC MYOCARDIAL CHANGE.	
epinephrin	401	TREATMENT OF	205
iodides	405	CHRONIC SYSTEMIC DISEASES.	36
morphine	404	CHRONIC VALVULAR DISEASE.	
strophanthus	399	See VALVULAR DISEASE .	242
strychnine	405	CICATRICAL MYOCARDITIS ...	195
the nitrites	402	CIRCULATION OF THE BLOOD ..	14
CARDIAC FLATNESS	59		

	PAGE		PAGE
CIRCULATORY EFFECTS OF ARTERIOSCLEROSIS	279	CONGENITAL HEART AFFECTIONS (<i>continued</i>)	
CLASSIFICATION OF PERICARDITIS	164	persistent ductus arteriosus	274
CLIMATE	377	valve defects	274
CLINICAL SIGNIFICANCE OF VALVULAR LESIONS	268	valvular disease, congenital	276
CLOTHING		varieties of	270
removal of, during examination	41	CONSTITUTIONAL INFERIORITY	354
selection of, for cardiopaths	371	CONSTRICITION, SENSE OF, IN ANGINA PECTORIS	338
"CLUBBING OF FINGER TIPS"	251	CONTEST, ELEMENT OF, UNDESIRABLE	376
COLLOIDAL GOLD TEST IN CARDIAC DIAGNOSIS	83	CONTRACTILITY	18
COHN, ALFRED E.	396	CONTRACTILITY AND PULSUS ALTERNANS	160
COMBINED ANEURISM	308	CONTRACTILITY DISTURBANCES IN ARTERIOSCLEROSIS	295
COMPARATIVE READINGS IN BLOOD-PRESSURE ESTIMATES	127	CONTRACTION IMPULSE, SPEED OF	17
COMPARATIVE READING OF ELECTROCARDIOGRAM	122	CONTRACTIONS, PREMATURE. See PREMATURE CONTRACTIONS	28
COMPARATIVE VALUES	7	CONUS ARTERIOSUS, MURMURS ARISING IN	79, 267
COMPENSATORY PAUSE	49	CONVALESCENCE	
COMPLETE DISSOCIATION, ATRIO-VENTRICULAR	157	from endocarditis	240
CONDUCTION DISTURBANCES IN ARTERIOSCLEROSIS	294	insufficient period of	34
CONDUCTION INTERVAL		CONVALESCENT PERIOD	34
of electrocardiogram	116	CORONARY ARTERIES	20
of polygram	97	COR BOVINUM	258
CONDUCTION SYSTEM	15	CORRADI'S METHOD IN ANEURISM	335
bundle of His	16	CORRIGAN'S "BUTTON HOLE MITRAL"	244
fibers of Purkinje	16	CORRIGAN'S DISEASE	254
in electrocardiography	103	CORRIGAN, DOMINIC	254
node of Tawara	16	CORRIGAN'S PULSE	258
pacemaker	15	COUGH	
sino-auricular node	15	unusual causes of	26
rates in various portions of	17, 18	as an evidence of heart affections	26
CONDUCTIVITY	18	CRESENDO MURMURS	75
CONGENITAL HEART AFFECTIONS	270	CROWNED TEETH	37
conclusions	277	"CUMULATIVE ACTION" OF DIGITALIS	153
defects of interauricular septum	270	CUSHING AND EDWARDS	145
dextrocardia	276	c WAVE	95
ductus Botalli	274	c WAVE OF PHLEBOGRAM	95
foramen ovale, patulous ...	270	CYANOSIS	28
semi-patulous	271	CYANOSIS DUE TO FORAMEN OVALE	12
perforate interventricular septum	274		

	PAGE		PAGE
CYCLE, CARDIAC	18	DIET IN HEART AFFECTIONS ..	384
CYCLING	376	DIET, KARELL'S	211
DA COSTA, J. M.	352	DIET, TUFNELL'S	333
"D. A. H." See NEURO-CIR- CULATORY ASTHENIA	352	DIFFERENTIAL BLOOD COUNTS IN CARDIAC DIAGNOSIS ..	83
DAILY LIFE OF PATIENT	370	DIFFERENTIATION OF MURMURS, FRENCH METHOD	80
DANCING	376	DIFFUSE ANEURISM	308
DARWIN, ON PALPITATION	25	DIGITALIS	394
DEATH, IMPENDING, IN AN- GINA PECTORIS	338, 344	cumulative action of	153
DECRESCENT TYPE OF ARTERIO- SCLEROSIS	290	frequent causes of heart- block	157
DECREASE IN HEART SIZE FOL- LOWING EXERCISE	67	in angina pectoris	350
DEFICIT OF PULSE	148	in arteriosclerosis	304
DEFINITION OF ENDOCARDITIS..	215	in endocarditis	240
DEFINITION OF HEART-BLOCK .	156	infusion of	209
DEGREES OF HEART-BLOCK	157	in heart-block	160
DELAYED CONDUCTION	157	in myocarditis	214
electrocardiogram of	115	thoughtful employment of.	154
"DELIRIUM OF HEART DIS- EASE"	262	variability of dose in auric- ular fibrillation	154
DELIRIUM CORDIS	145	used by malingerers	415
DERMOGRAPHIA	257	DILATATION OF AORTA	311
DEXTROCARDIA	276	DIMINUENDO MURMURS	75
a rare condition	66	DIPHASIC WAVES	
changes usual percussion note	66	definition of	122
in electrocardiogram	123	DIPHThERIA	34
d NOTCH OF ARTERIOGRAM ...	5	DIPHThERIA AND MYOCARDITIS	190
DEFICIT OF PULSE	48	DISSEMBLING	412
DEEP CARDIAC DULLNESS	59	DISEASES OF CHILDHOOD IN MYOCARDITIS	190
DIAGNOSIS		"DISORDERED ACTION OF THE HEART"	352
instrumental methods of ..	2	DISSECTING ANEURISM	307
DIAMETERS, CARDIAC	68	DISSOCIATION, COMPLETE A-V	157
DIAMETERS, CARDIAC, IN MITRAL STENOSIS	259	DISTRESSING INCIDENCES DUR- ING MYOCARDITIS	213
DIASTOLE		DIURETIC R	302
of arteriogram	95	DIZZINESS	25
of electrocardiogram	121	DOLORME AND MIGNON, STA- TISTICS OF	188
DIASTOLIC MURMUR	259	DOMINANT RHYTHM	98
DIASTOLIC PERIOD OF ELECTRO- CARDIOGRAM	121	"DON'TS IN BLOOD-PRESSURE .	130
DIASTOLIC SHOCK OF ANEU- RISM	315	DROPPED BEATS, ACTUAL. 28, 49, 157	
DICROTIC NOTCH	95	electrocardiogram of	116
unusual depth of	101	polygram of	99
DICROTIC PULSE	49	"DROPPED HEART"	5
		"DROPPING OF THE HEART BEAT"	138
		DRUGS, CARDIAC	393

	PAGE		PAGE
DRUGS IN MYOCARDITIS	214	ELECTROCARDIOGRAM (<i>continued</i>)	
DURATION OF MURMURS	75	auricular fibrillation	115
DUCTLESS GLANDS AND ARTERIOSCLEROSIS	285	flutter	114
DUCTUS ARTERIOSUS, PERSISTENT	274	comparative reading	122
DUCTUS BOTALLI	274	component parts	111
DUROZIEZ'S SIGN	260	definition of	103
DYSPNŒA	26	delayed conduction	115
DYSPNŒA, DUE TO PERSISTENT FORAMEN OVALE ...	17	dextrocardia	123
EAR DISEASE	37	diastolic period	121
EARLY FATIGUE	24	diphasic waves	122
EARLY SYMPTOMS OF ARTERIOSCLEROSIS	288	dropped beat	116
EARLY SYMPTOMS OF HEART AFFECTIONS	23	general survey	122
EARLY TO BED, VALUE OF	374	heart sounds in	118, 121
EARLY RECOGNITION OF HEART AFFECTIONS	363	high grade block	117
ECTOPIA CORDIS ABDOMINALIS.	65	importance of Lead II	122
EDEMA		leads, definition of	110
relief of	213	lines of the record	112
scrotal	28	mitral stenosis	119
EDWARDS AND CUSHING	145	myocarditis	200
EFFLEURAGE, EMPLOYMENT OF.	387	normal	110
EFFORT, IN HEART PATIENTS	371	normal record	111
"EFFORT SYNDROME." See NEURO-CIRCULATORY ASTHENIA	37	ordinates	112
EFFUSION <i>vs.</i> EXPLORATORY PUNCTURE, IN PERICARDITIS	178	premature contractions	113
EFFUSION, PERICARDIAL <i>vs.</i> CARDIAC ENLARGEMENT ..	176	principle of	103
EFFUSION, PERICARDIAL <i>vs.</i> PLEURAL	176	P-R interval	116
EFFUSION, PERICARDITIS WITH.	172	P wave	114
EFFUSION, SIGNS OF PERICARDIAL	174	Q-R-S-T complex	116
EINTHOVEN, W.	104	Q wave	116
ELECTRICITY IN ARTERIOSCLEROSIS	306	R wave	118
ELECTROCARDIOGRAM	102	second lead reading	122
abscissæ	112	sinus arrhythmia	112
arborization block ..	118	suggestions in analyzing ...	123
artefacts	122	S wave	120
auricular enlargement	119	symbols of	112
		tachycardia	113
		T wave	120
		U wave	121
		ventricular preponderance .	120
		ELECTROCARDIOGRAPH	104
		contribution to science ...	2
		description of	104
		devised by Einthoven	104
		purpose of	2
		ELECTROCARDIOGRAPHY	125
		abuse of	125
		compared with other laboratory aids	102
		diagnoses otherwise not possible	109
		electrodes	112
		experimental investigations.	106
		first American papers	103
		questions the physician asks.	106
		solution for electrodes ...	112
		use of	125

	PAGE		PAGE
ELECTRODES, ELECTROCARDIO-GRAPHIC		EXAMINATION	
composition of	112	general considerations	21
solution for	112	graphic methods of	89
ELEMENT OF CONTEST, UN-DESIRABLE	376	EXAMINATION OF PATIENT	
ELIMINATION IN HEART AFFECTIONS	301	auscultation	70
ELIMINATION IN MYOCARDITIS.	208	blood-pressure estimates ..	127
EMBOLIC ANEURISM	308	diagnostic points	22
EMBOLISM	233	general considerations	21
EMBOLISM, PARADOXICAL	273	graphic methods, the elec-	
EMOTIONAL DISTURBANCES, AVOIDANCE OF, IN MYOCARDITIS	208	trocardiogram	103
EMOTIONAL STRESS	37	the polygram	89
EMPHYSEMA OF VON BASCH ..	345	habits	38
ENDARTERITIS OBLITERANS ...	278	inspection	42
ENDOCARDITIS	215	laboratory aids	82
acute, diagnosis of	232	mensuration	67
etiology of	221	palpation	44
morbid anatomy of	217	palpation of pulse	47
physical signs of	230	percussion	58
prognosis of	235	preparation	41
symptoms of	229	presenting symptoms	23
a secondary process	216	previous history	34
convalescence from	240	pulse rate	49
definition of	215	rate response to exercise ..	51
focal infections as a cause of	224	rhythm of pulse	55
gonorrheal	35	volume of pulse	56
malignant, diagnosis of ...	234	"EXCITABLE HEART." See	
emaciation in	236	NEURO-CIRCULATORY AS-	
etiology of	227	THENIA	352
morbid anatomy of	219	EXCITABILITY OF HEART MUS-	
prognosis of	236	CLE	18
symptoms of	233	EXCITATION WAVE	16
not confined to valves alone.	215	in electrocardiography	103
streptococcus viridans in ..	216	EXERCISE	372
treatment of	236	contraindications	54
uterine life, during	219	decreases size of normal	
varieties of	216	heart	67
verrucose	217	error, sources of	55
EPINEPHRIN	341	graded	383
EPINEPHRIN IN HEART-BLOCK.	160	gradual return to	383
"EROSION," ANEURISMAL	323	hopping test	51
ERYSIPELAS, BLOCK OCCURRING DURING	156	interdicted in acute condi-	
ETIOLOGY OF ACUTE ENDOCARDITIS	221	tions	51
ETIOLOGY OF MALIGNANT ENDOCARDITIS	227	influence on murmurs	77
ETHER	411	in pericarditis	186
		rate response, importance	
		of	51
		resistance exercises	384
		various forms of	51
		EXPERIMENTS OF PILCHER AND	
		SOLLMAN	407
		EXPERIMENTS OF STEWART AND	
		ROGOLF	401
		"EXTRA SYSTOLES," OBJEC-	
		TIONABLE TERM	136

	PAGE		PAGE
EYE SIGNS IN ARTERIOSCLE- ROSIS	295	GALL-BLADDER INFECTIONS ...	37
FACTORS IN BLOOD-PRESSURE ..	131	GALLOP RHYTHM in heart-block	158
FAINTING	27	production of	77
FALSE ANEURISM	307	GALVANOMETER, STRING	105
FATIGUE early, importance of	24	GARROD, A. E.	167
FATTY DEGENERATION	196	"GAS, PRESSURE OF"	385
"FATTY" HEART	196	GENERAL CONSIDERATIONS OF VALVULAR DISEASE	242
FATTY INFILTRATION	196	GENERAL SURVEY OF ELECTRO- CARDIOGRAM	122
FAULTY DENTISTRY	36	GENUINE MENTAL ATMOS- PHERES	417
FEBRILE MURMURS	79	GERMAN SPAS	388
FEVER, EFFECT OF	35	GERMAN SPA TREATMENT ...	359
FIBRILLATION. See AURICULAR FIBRILLATION	145	GIDDINESS	25
FIBRILLATION TRIAD	149	GILLESPIE, STATISTICS OF	248
FIBROUS MYOCARDITIS	202	GOITER	36
FISHING, AS EXERCISE FOR CARDIOPATHS	374	von Grafe's sign	43
FOREIGN BODY IN HEART	365	GOLF, FOR CARDIOPATHS	376
"FUNCTIONAL HEART DIS- EASE." See NEURO-CIRCU- LATORY ASTHENIA	352	GOULSTON, SIR ARTHUR	211
FLAT BRANCH OF BUNDLE ...	16	GONORRHEAL ENDOCARDITIS ..	35
FLINT MURMUR	253	"GOOSE NECK" ARTERIES	294
FLUOROSCOPE IN CARDIAC DIAGNOSIS	86	GOUT	36
FOCAL INFECTION	36	GRADED EXERCISE	383
FOCAL INFECTIONS AND ARTE- RIOSCLEROSIS	284	GRADES OF HEART-BLOCK	157
FOCAL INFECTIONS AS CAUSE OF ENDOCARDITIS	224	GRAPHIC METHODS OF EXAM- INATION	99
FOWLER'S SOLUTION IN ENDO- CARDITIS	238	GRAPHIC RECORDS normal electrocardiogram .	111
FORAMEN OVALE, PATULOUS .	270	polygram	95
FORD STETHOSCOPE	71	"GROWING PAINS" OF CHILD- HOOD	221
FORMS for cardiovascular rec- ords	31, 32	GYMNASTIC TREATMENT OF HEART DISEASE	384
FRENCH METHOD OF DIFFEREN- TIATING MURMURS	80	HABITS alcohol	38
FREQUENCY OF VALVULAR LES- IONS	246	"athletes' heart"	39
FRICTION, PERICARDIAL	170	excessive physical exercise.	39
FUNCTIONAL BLOCK	156	habit forming drugs	40
FUNK, ELMER H.	330	tobacco	38
"FUNNEL-SHAPED MITRAL" ..	244	HARVEY, WILLIAM	14
		HEART "adolescent"	37
		anatomy of	4
		"athletes"	39
		auscultation of	70
		average measurements of .	5
		capacity of structure	12
		chambers of	7

	PAGE		PAGE
HEART (<i>continued</i>).		HEART MUSCLE WEARINESS,	
cycle of	18	SYMPTOMS OF	23
emotional stress	37	HEART NUTRITION DURING	
febrile murmurs	79	MYOCARDITIS	210
internal architectures of. 7, 8, 9		HEART RATE	
left border of	59	measurements of, in elec-	
malposition of	64	trocardiogram	122
murmurs of	74	in polygram	95
without significance	78	HEART SOUNDS SYNCHRON-	
natural sounds of	72	OUS WITH ELECTROCARDIO-	
nerves of	19	GRAM	118, 121
"neurotic"	37	HEART SOUNDS IN MYOCAR-	
oppression of, during sleep.	27	DITIS	199
pacemaker of	15	HEART SOUNDS IN NEURO-CIR-	
physical strain	37	CULATORY ASTHENIA	358
preliminary rules of study.	3	HEMOPERICARDIUM	166
presenting symptoms	23	"HEMIC" MURMURS	257
properties of muscle	18	HERRICK, JAMES	265, 330
puncta maxima	73	HIGH GRADE HEART-BLOCK ..	157
ratio of work and rest	15	HILL, LEONARD	261
relation to chest wall	6	HONAN, JAMES	347
right border of	60	HORSEBACK RIDING FOR CAR-	
shape	4	DIOPATHS	377
significant murmurs	80	HOWARD'S STATISTICS OF AN-	
size	4	EURISM	316
size decreases on exercise .	67	h WAVE	96
"soldier's heart"	63	HYDROPERICARDIUM	166
systole of	15	HYPERESTHESIA, CUTANEOUS .	45
thrills	44	HYPERPIESIA	131
tobacco, influence of	39	HYPERTENSION	
valves of	11	abdominal support in	133
weight of	4	drugs in	133
HEART AFFECTIONS, CONGENI-		physiologic	132
TAL	270	venesection in	133
HEART-BLOCK. See "BLOCK".	156	treatment of	132
auricular activity in	159	HYPOTENSION	131
complete	157	HYPERTHYROIDISM	140
rates suggestive of	50	HYSTERIA	339
HEART-BLOCK, HIGH GRADE ..	157		
electrocardiogram of	117	INDUCTION OF CHRONIC MYO-	
complete in polygram	101	CARDIAL CHANGE	194
polygram of	99	INFECTIONS	
recognition in polygram ..	99	dental	37
HEART BORDERS IN NEURO-CIR-		focal	36
CULATORY ASTHENIA	358	of adolescence	35
HEART DISEASE		of appendix	37
a term of terror	29, 30	of childhood	34
evolution of	34	of gall-bladder	37
"inherited"	40	oral	37
"HEART DISEASE DELIRIUM" .	262	septic absorption	36
HEART FAILURE		systemic diseases	36
advanced signs of	202		
early symptoms of	23		
HEART IN ARTERIOSCLEROSIS ..	292		
HEART MUSCLE, STRENGTH OF.	12		

	PAGE		PAGE
INFECTIONS, ACUTE AND ARTERIOSCLEROSIS	284	IRREGULAR PULSE (<i>continued</i>).	
INFECTIONS OF CHILDHOOD ...	366	auricular flutter	144
INFILTRATION OF ARTERIES ...	294	clinical significance of ...	135
INFLUENCES		general considerations	134
affecting the heart	5	heart-block	156
vagal	20	often foreshadows heart	
INFLUENZA	35	failure	163
INFLUENZA AND MYOCARDITIS ..	190	paroxysmal tachycardia ...	139
INFUSION OF DIGITALIS	209	premature contractions ...	136
"INHERITED HEART DISEASE" ..	40	pulsus alternans	160
INHIBITORY NERVES	19	sinus arrhythmia	135
INSOMNIA IN ENDOCARDITIS ..	239	IRRITABLE HEART OF SOLDIERS.	
INSTRUCTION OF PUBLIC IN		See NEURO-CIRCULATORY	
HEART AFFECTIONS	362	ASTHENIA	352
INSUFFICIENCY OF AORTIC		ISOELECTRIC LINE	120
VALVE	254	JANEWAY, THEODORE	284
INSUFFICIENCY OF MITRAL		JUGULAR	
VALVE	248	cup, of polygraph	93
INSUFFICIENCY OF PULMONARY		a-c-v waves of tracing	95
VALVE	267	pulse, recording of	93
INSUFFICIENCY OF TRICUSPID		tracing	95
VALVE	264	JUGULAR WAVES, VARIATIONS	
INSUFFICIENCY OF VALVES ...	246	IN	96
INSUFFICIENT CONVALESCENCE.	34	KARELL DIET	211
INTENSITY OF MURMURS	75	KAROTKOW, AUSCULTATORY	
INTERAURICULAR SEPTUM	12	METHOD OF	129
INTERMITTENT CLAUDICATION ..	289	KEEPING OF RECORDS	30
INTERPRETATION OF ARTERIO-		KEITH, ARTHUR	14
GRAM	97	KUSSMAULL'S SIGN	183
INTERPRETATION OF ELECTRO-		LABILITY OF PULSE	139
CARDIOGRAM	102	LABORATORY AIDS IN DIAG-	
INTERPRETATION OF PHLEBO-		NOSIS	82
GRAM	95	blood cultures	83
INTERPRETING THE POLYGRAM.	97	colloidal gold test	83
INTERVENTRICULAR SEPTUM,		differential blood counts ..	83
DEFECTS OF	274	fluoroscope	86
INTIMA OF ARTERIES	279	leukocytosis	83
INSTRUMENTAL METHODS,		lymphocytosis	83
PLACE OF	2	ophthalmoscope	82
IODIDE OF POTASH IN ARTERIO-		orthodiascope	86
SCLEROSIS	305	renal function test	82
IODIDES	405	skiagraph	86
IODISM	305	urinalysis	82
IRRADIATION	43, 230	x-ray	86
IRRADIATION IN NEURO-CIRCU-		Wassermann reaction	83
LATORY ASTHENIA	357	LAUGHING GAS	411
IRREGULAR PULSE	134	"LEAKING VALVES"	242
auricular fibrillation	145	"LEATHERY" FRICTION RUB ...	170
		LEFT BORDER OF HEART	59
		location by maximum im-	
		pulse	59

	PAGE		PAGE
LEFT HEART	7	MECHANISM, CARDIAC, ABNORMALITIES OF. See IRREGULAR PULSE	124
LEMANN, STATISTICS OF, IN ANEURISM	328	MEDIA	279
LEUKOCYTOSIS IN CARDIAC DIAGNOSIS	83	MEDIASTINO-PERICARDITIS	180
LEWIS, THOMAS	15-47	MEDIASTINAL TUMORS	66
LIGAMENTUM ARTERIOSUM ...	274	MENIERE'S DISEASE	25
LINES		MENSURATION	
mid-sternal	67	mid-sternal line	67
on electrocardiogram	112	usual measurements of	
LOCATION OF HEART-BLOCK ...	156	adults	68
LOCKE, F. S.	211	of children	69
LOCOMOTOR ARTERIES	294	technique of	68
LONELINESS, EFFECT OF	37	MENTAL REST IN MYOCARDITIS.	207
LOW GRADE HEART-BLOCK	157	MID-STERNAL LINE	67
LYMPH GLANDS AND VAGAL PRESSURE	142	MIGNON AND DOLORME, STATISTICS OF	188
LYMPHOCYTOSIS IN CARDIAC DIAGNOSIS	83	MILITARY LIFE, EFFECT OF ..	353
"M ¹ + "	77	MINERAL POISONS	36
MACKENZIE INK POLYGRAPH .	89	MINERAL WATERS	209
MACKENZIE, JAMES, AND MODERN CARDIOLOGY	362	MITRAL INSUFFICIENCY	248
MACKENZIE, SIR JAMES	47	group of essential signs ...	249
MALFORMATIONS OF THE CHEST	65	incorrectly diagnosed	248
MALINGERING	412	MITRAL REGURGITATION. See	
MALPOSITION OF THE HEART ..	64	MITRAL INSUFFICIENCY ..	248
MALPOSITION OF THE PATIENT.	64	MITRAL STENOSIS	249
MARRIAGE	377	apical presystolic murmur .	252
"MASSACRE OF THE TONSIL" .	226	crescendo murmur	252
MASSAGE	386	diameter of heart	254
MASSAGE, RATIONAL OF	387	duration of murmur	251
"MASTURBATOR'S HEART." See		electrocardiogram from ..	119
NEURO-CIRCULATORY ASTHENIA	352	essential signs	250
"MATTRESS OF FIBRIN" IN ANEURISM	315	presystolic thrill	253
MAXIMUM CARDIAC IMPULSE. .	43	"snappy" first sound	252
MEASLES	34	with auricular fibrillation ..	250
MEASLES, POSSIBLE RELATION OF, TO MYOCARDITIS	190	with insufficiency	254
MEASUREMENTS OF HEART ...	68	MITRAL VALVE	
MEASUREMENTS OF PULSE RATES, IN ELECTROCARDIOGRAM	122	disease of	11
in polygram	95	MODERN PROCEDURES IN HEART AFFECTIONS	364
MECHANICAL IRRITATION AND ARTERIOSCLEROSIS	283	MORBID ANATOMY OF ACUTE ENDOCARDITIS	217
		MORBID ANATOMY OF MALIGNANT ENDOCARDITIS	219
		MORPHINE	404
		MORPHINE IN ENDOCARDITIS .	239
		MOTORING FOR HEART PATIENTS	376
		MURMURS	
		accentuations, associated ..	76
		arising in conus arteriosus.	79, 267

	PAGE		PAGE
MURMURS (<i>continued</i>).		MYOCARDITIS (<i>continued</i>).	
basal	79	distressing incidences dur-	
cardio-respiratory	78	ing course of	213
correlation with other evi-		diseases of childhood in ..	190
dence	81	drugs in	214
crescendo	75	electrocardiographic recog-	
definition of	74	nition of	200
designation of	75	elimination in	208
diastolic	75, 80, 259	emotional disturbances,	
differentiation of, French		avoidance of, during	208
method	80	etiology of acute	189
diminished tonicity	79	fatty degeneration	196
diminuendo	75	infiltration	196
duration	75	heart nutrition during	210
exercise, influence of	77	induction of chronic myo-	
febrile	79	cardial change	194
Flint	253	mental rest in	207
in acute endocarditis	229	pain in	198
in neuro-circulatory as-		physical rest in	206
thenia	358	pulmonary valves in	198
intensity	75	pulse irregularities	204
of diagnostic import	80	terms employed	189
pitch	75	MYOCARDIUM, IMPORTANCE OF.	3
postural	79	MYOGENIC THEORY	18
presystolic	75, 80		
prolongation of first sound.	79	NATURAL HEART SOUNDS	72
quality	75	NAUHEIM BATHS	389
reduplications	77	NEGATIVE WAVES	121
requirements of	81	NEOARSPHENAMINE	404
significance of	3, 7, 14	NEOSALVARSAN	404
systolic	75	NEPHRITIS	36
tonal qualities of	75	NERVES, CARDIAC	19, 20
without significance	78	"NERVOUS HEART." See NEU-	
MURMURS WITHOUT SIGNIFI-		RO-CIRCULATORY AS-	
CANCE		THENIA	352
arising in conus arteriosus.	79	NEURO-CIRCULATORY ASTHE-	
basal	79	NIA	352
cardio-respiratory	78	definition of	352
diminished tonicity	79	in civil life	352
postural	79	in training camps	353
prolongation of first sound.	79	in war	354
statistics of	78	points in analysis	356
MYOCARDIAL AFFECTIONS	189	predisposing conditions ...	354
MYOCARDITIS	189	sinus arrhythmia in	135
acute myocarditis, diagnosis		treatment of	359
of	197	typical case record	355
treatment of	204	NEVUS VASCULOSIS	307
advanced myocardial dam-		NITRITES IN ARTERIOSCLEROSIS.	304
age, signs of	202	NITROGLYCERIN IN ANGINA	
breathing exercises in	212	PECTORIS	348
cardiosclerosis	196	NITROUS OXIDE	411
chronic myocardial affec-		NORMAL ARTERIOGRAM	95
tions, etiology of	193		
change, recognition of ..	201		
treatment of	205		

	PAGE		PAGE
NUTRITION		PALPITATION	25
influence on heart	5	PANCARDITIS	220
OBJECTIONABLE TERMS		PARACENTESIS PERICARDII. 184, 187	
anterior axillary line	67	PAROXYSMAL TACHYCARDIA ..	139
"cumulative action" of digi-		auricular fibrillation	140
talis	150	flutter	140
"extra systoles"	136	causes of	139
"false angina"	338	electrocardiogram of	113
"fatty" heart	196	hyperthyroidism	140
"gastric flatulence"	24	ordinary tachycardia	39
heart disease	30	prognosis of	141
"inherited" heart disease ..	40	treatment of	141
"intercostal neuralgia" ...	24	true paroxysmal tachycar-	
mid-clavicular line	67	dia	140
"mock angina"	338	PATIENT	
nipple line	67	attitude of	29
"pseudo angina"	338	examination of	21
"rheumatism"	35	first observations of	21
"rheumatism of the heart" .	217	general considerations ...	21
OBLITERATION OF RADIAL		malposition of	62
PULSE	316	position of	42
OCULO-CARDIAC REFLEX	80	preparation of	41
OCCUPATION		previous history	34
effect on heart	5	PATULOUS FORAMEN OVALE ..	270
OERTEL'S TREATMENT	268	PEARLS OF AMYL NITRITE ...	403
OLIVER'S SIGN	45, 323	PERICARDIAL ADHESIONS	65
OPHTHALMOSCOPE IN ARTERIO-		PERICARDIAL EFFUSIONS	66
SCLEROSIS	296	PERICARDIAL SAC, CAPACITY	
OPHTHALMOSCOPE IN CARDIAC		OF	172
DIAGNOSIS	82	"PERICARDITIC PSEUDOCIRRH-	
OPPRESSION DURING SLEEP ..	27	SIS OF THE LIVER"	184
ORDINATES		PERICARDITIS	
of electrocardiogram	112	acute	167
of polygram	91	adhesions, extra-pericardial.	179
ORTHODIAGRAPH		intra-pericardial	179
purpose of	2	physical signs of	180
ORTHODIASCOPE IN CARDIAC		adherent pericardium	182
DIAGNOSIS	86	classification of	164
ORTHOPNEA	27	effusion, signs of	174
OVEREATING AND ARTERIOSCLE-		exploratory puncture in .	178
ROSIS	283	hemopericardium	166
Ox, AORTIC VALVE OF	11	hydropericardium	166
"Ox HEART"	258	paracentesis in	187
"P2 + "	76	pericardial <i>vs.</i> pleural effu-	
PACEMAKER	15	sions	176
PAIN, ANGINAL	337, 343	pericardial effusions <i>vs.</i> car-	
PAIN, ARTERIAL	340	diac enlargement	176
PAIN IN MYOCARDITIS	198	Pick's syndrome	184
PAIN IN PERICARDITIS	171	pneumopericardium	166
		pulmonary compression	
		signs in	171
		respiratory rate in	172
		treatment of	184

	PAGE		PAGE
PERICARDITIS (<i>continued</i>).		POINT OF MAXIMUM INTEN-	
with effusion	172	SITY	45
PERICARDIUM, SIGNIFICANCE		POLYCYTHEMIA	28, 83
OF	164	POLYGRAM	
PERIPHERAL CIRCULATION IN		abuse of	101
ANEURISM	316	apparatus for recording ...	89
PERCUSSION, CARDIAC	58	auricular flutter in	101
cardiac flatness	59	cardiogram	91
definition of	58	component parts of tracing.	90
deep cardiac dullness	59	interpretation of	97
forms of	58	ordinates	91
importance of	58	making of tracings	94
left border of heart	59	normal arteriogram	95
malposition, changes in-		phlebogram	95
duced by	62	sentence suggestions in an-	
method of	59	alysis	100
purpose of	58	technique	92
resistance, sense of	58	time record	91
right border of heart	60	POLYGRAPH	
sources of error in	58	advantages of	89
superficial cardiac dullness.	59	apparatus	89
transverse diameter	60	contribution to science ..	2
PERCUSSION WAVE	95	definition of	89
PHLEBOGRAM	91	operation of	92
<i>a</i> Wave	95	purpose of	2, 89
<i>c</i> Wave	95	the jugular cup	93
<i>v</i> Wave	95	the wrist piece	92
identification of waves ...	95	POSITION OF PATIENT	42
PHILLIP, W. F. R.	271	POSITIVE WAVES	121
PHYSICAL REST IN MYOCAR-		POSTERIOR AURICLE	10
DITIS	206	POSTURAL MURMURS	79
PHYSICAL SIGNS OF ACUTE		POSTURE, CHANGE OF	383
ENDOCARDITIS	230	POTASSIUM IODIDE, REASONS	
PHYSICAL STRAIN	37	FOR EFFECTIVENESS	305
PHYSIOLOGY		PRATT, JOSEPH	399
of rest	381	PRECORDIAL	
of the heart	4	hyperesthesia	24
PICK'S SYNDROME	184	impulse	43
PILCHER AND SOLLMAN, Ex-		oppression, early, impor-	
PERIMENTS OF	407	tance of	24
PITCH OF MURMURS	75	pain	24
"PIPE STEM" ARTERIES	294	thrills	44
"PISTOL SHOT FEMORAL"	260	PRECORDIAL OPPRESSION IN	
"PISTOL SHOT" PULSE	258	MYOCARDITIS	198
PLAQUES, ATHEROMATOUS ...	278	PRECORDIAL PAIN IN MYO-	
PLEURAL EFFUSIONS	65	CARDITIS	198
PLEXUS CARDIAC	20	PRECORDIAL PAIN IN NEURO-	
PNEUMOGASTRIC NERVE		CIRCULATORY ASTHENIA ..	356
distribution of fibers	19	PREGNANCY	379
PNEUMONIA	35	<i>P-R</i> INTERVAL	116
PNEUMOPERICARDIUM	166	PRELIMINARY RULES IN HEART	
		STUDY	3
		PRELIMINARY INSPECTION	42

	PAGE		PAGE
PREMATURE CONTRACTIONS. 28, 49		PROPHYLAXIS OF HEART DIS-	
alternation following	100	EASE	369
auricular in polygram	99	PUBLIC INSTRUCTION IN HEART	
compensatory pause	136	AFFECTIONS	362
definition of	136	PULMONARY ARTERY, ABSENCE	
electrocardiogram of	113	OF	277
in electrocardiogram	123	PULMONARY COMPRESSION	
polygram of	100	SIGNS IN PERICARDITIS ..	171
significance of	138	PULMONARY INSUFFICIENCY,	
ventricular, in polygram ..	99	PHYSICAL SIGNS OF	267
ventricular type more fre-		PULMONARY STENOSIS, CON-	
quent	137	GENITAL	276
<i>vs.</i> actual dropped beats ..	158	PULMONARY STENOSIS, PHYS-	
<i>vs.</i> bigeminy	101	ICAL SIGNS OF	266
PREPARATION OF THE PATIENT. 41		PULMONARY VALVES IN MYO-	
PRE-SCLEROTIC STAGE OF AR-		CARDITIS	198
TERIOSCLEROSIS	288	PULSATIONS IN NEURO-CIRC-	
PRESENTING SYMPTOMS		LATORY ASTHENIA	357
cardiac sleep start	27	PULSE	
Cheyne-Stokes breathing ..	29	bimanual estimates	48
cough	26	condition of arteries	48
cyanosis	28	Corrigan's	258
dropped beats	28	deficit of	48
dyspnea	26	dicrotic	40
early exhaustion	24	five-second grouping	50
edema	28	in acute endocarditis	230
fainting	28	in arteriosclerosis	293
giddiness	25	influence of tobacco on ...	39
palpitation	25	in neuro-circulatory asthe-	
precordial hyperesthesia ..	24	nia	357
oppression	24	lability of	139
pain	24	method of counting	50
table of	23	natural rate	50
"PRESSURE OF GAS"	385	palpation of	47
PRESYSTOLIC MURMUR IN MIT-		"Pistol shot"	258
RAL STENOSIS	252	pulse-rate	49
PRESYSTOLIC THRILL	253	rate response	51
PREVENTION OF HEART AFFEC-		reasons for examining	47
TIONS	366	retardation of	48
PREVIOUS DISEASE		rhythm	55
influence on heart	5	slow	50
PREVIOUS HISTORY		technique	47
importance of	34	volume	56
PRIMARY ANEURISMS	307	PULSE DEFICIT	148
PROGNOSIS OF ACUTE ENDO-		PULSE IRREGULARITIES. See	
CARDITIS	235	IRREGULAR PULSE	134
PROGNOSIS OF HEART-BLOCK ..	159	PULSE IRREGULARITIES IN MY-	
PROGNOSIS OF MALIGNANT EN-		OCARDITIS	204
DOCARDITIS	236	PULSE IRREGULARITIES OF	
PROLONGATION OF FIRST		HEART FAILURE	204
SOUND	79	PULSE PERIOD	19
PROPERTIES OF HEART MUSCLE. 18		PULSE, RADIAL, OBLITERATION	
		OF	319

	PAGE		PAGE
PULSE, "WATER-HAMMER" . . .	258	RECOGNITION OF CHRONIC MY-	
PULSUS ALTERNANS	160	OCARDIAL CHANGE	201
alternation must alternate .	161	RECORDS	
contractility and pulsus al-		the keeping of	30
ternans	160	cardiovascular blank	31, 32
definition of	160	REDUPLICATIONS	77
detection of	57	"REGION OF CARDIAC ROM-	
diagnosis of	161	ANCE"	267
following premature con-		REGURGITATION, MITRAL	248
tractions	161	REGURGITATION, PULMONARY .	267
significance of	161	REGURGITATION, TRICUSPID . .	264
transient pulsus alternans .	161	RELAXATION, IMPORTANCE OF .	299
in polygram	100	REQUIREMENTS OF MURMURS .	81
method of eliciting	57	RESISTANCE EXERCISES	384
PULSUS ARRHYTHMIAS	145	REST, IN ARTERIOSCLEROSIS . .	300
PULSUS IRREGULARIS PER-		REST, PHYSIOLOGY OF	381
PETUUS	145	REST, THERAPY OF	381
PURKINJE	16	RESUMPTION OF ACTIVITIES . .	383
fibers, block of	156	RETINA IN ARTERIOSCLEROSIS .	295
p WAVE	95-114	RENAL FUNCTION IN CARDIAC	
p WAVE OF ARTERIOGRAM . . .	95	DIAGNOSIS	82
PYORRHEA	37	RESPIRATION	
Q-R-S-T COMPLEX	116	Cheyne-Stokes	29
QUALITY OF MURMURS	75	RESPIRATORY RATE IN PERI-	
QUINCKE'S CAPILLARY PULSE.	257	CARDITIS	72
Q WAVE	116	REST AND WORK, RATIO OF . .	15
RATES		REST, INSUFFICIENT, AND AR-	
cardiac, following exer-		TERIOSCLEROSIS	282
cise	51, 53	REST PERIOD FOLLOWING EN-	
respiratory following exer-		DOCARDITIS	240
cise	51, 54	RETRACTION OF VALVES	244
RATE DISTURBANCE IN ARTE-		RETARDATION OF PULSE	48
RIOSCLEROSIS	294	RHEUMATIC FEVER	35
RATES OF BUNDLE OF HIS . . .	18	"RHEUMATIC NODULES"	234
RATE OF PULSE	49	"RHEUMATISM"	
observation in medical		an objectionable term	35
corps	50	RHYTHM	55
RATE RESPONSE TO EXERCISE		bimanual estimation of . . .	56
contra-indications of exer-		coupled beats	56
cise	54	definition of	55
electrocardiogram of	52	digitalis coupling	56
error, sources of	55	disturbances in arterioscle-	
hopping test	51	rosis	294
importance of	51	dominant	98
in neuro-circulatory asthe-		five-second count	56
nia	358	triple beats	56
natural rate response	53	RIGHT BORDER OF HEART	60
table of	53	RIGHT HEART	7
various forms of	51	ROBEY, WILLIAM H., JR. . . .	167
RATIO OF WORK AND REST OF		ROUND BUNDLE BRANCH	16
HEART	15		

	PAGE		PAGE
ROWING	375	SLEEP	
RUBBING	387	oppression of heart during .	27
RULES FOR ANALYZING ELEC-		SLEEP START, CARDIAC	27
TROCARDIOGRAMS	122	SMITH, ALLEN J. ...	285, 315, 323
RUPTURE OF ANEURISM	328	SOCIAL AFFAIRS FOR CARDIO-	
R WAVE	118	PATHS	373
SACCULATED ANEURISM	307	SODIUM BICARBONATE IN EN-	
"SAFETY VALVE REGURGITATION"		DOCARDITIS	238
.....	264	SODIUM CACODYLATE IN PERI-	
SAJOUS, CHARLES E. DE M. ...	406	CARDITIS	186
SALVARSAN	404	SODIUM NITRITE IN ANGINA	
SANATORIUM TREATMENT. 207,	388	PECTORIS	350
SCARLET FEVER	34	"SOLDIER'S HEART"	63
SCHOTT, GYMNAS TIC TREAT-		SOUNDS	
MENT	384	abnormal, location of	74
SECOND LEAD READING OF		accentuations of	76
ELECTROCARDIOGRAM	122	arterial	76
SECONDARY ANEURISMS	307	artificial production of ...	76
SENECA'S DESCRIPTION OF AN-		causes of	73
GINA PECTORIS	343	first sound, qualities of ...	73
SENILE HEART, DIET FOR	386	French method of differen-	
SEPTUM, INTER-AURICULAR,		tiating	80
DEFECTS OF	270	friction rubs	77
SEX		natural, of heart	72
influence on heart	5	of heart in electrocardio-	
SEWALL, HENRY	130	gram	118, 121
SIGNIFICANCE OF CARDIAC IR-		on chest	72
REGULARITIES	162	puncta maxima	73
SIGNIFICANCE OF PERICAR-		reduplications of	77
DIUM	164	second sound, qualities of .	73
SIGNS IN ADVANCED MYOCAR-		split first sound	77
DIAL DAMAGE	202	transmission of	76
"SILVER WIRE" ARTERIES ...	296	without significance	78
SIMPLE ANEURISM	308	SOUTHEY'S TUBES	213
SIMULATING SOUNDS	266	SPA TREATMENT	359
SINO-AURICULAR BLOCK	156	SPEED OF CONTRACTION IM-	
SINO-AURICULAR NODE	15	PULSE	17
SINUS ARRHYTHMIA		SPINAL DEFORMITIES	65
electrocardiogram of	112	SPINE, PROXIMITY OF AORTA	
frequency in neuro-circula-		TO	263
tory asthenia	135	SPORTS	374
in polygram	100	"SPLIT FIRST SOUND"	77
not pathologic	136	STATURE	
polygram of	98	influence on heart	5
requires no treatment	136	START DURING SLEEP	27
SKATING	375	STENOCARDIA. See ANGINA	
SKIAGRAPH IN CARDIAC DIAG-		PECTORIS	338
NOSIS	86	STENOSIS OF AORTIC VALVE ..	262
		STENOSIS OF MITRAL VALVE ..	249
		STENOSIS OF PULMONARY	
		VALVE	266

INDEX.

437

	PAGE		PAGE
STENOSIS OF TRICUSPID VALVE	265	SYSTEMIC DISEASES, CHRONIC.	36
STENOSIS OF VALVES	246	SYSTOLE OF HEART	15
STETHOSCOPES		SYSTOLIC BRUITS, CAUSE OF	259
Ford	71	SYSTOLIC MURMURS AT BASE	259
requirements of	71	SWIMMING	375
varieties of	72		
STEWART AND ROGOLF, EXPERI- MENTS OF	401	TABAGISM AND ARTERIOSCLE- ROSIS	282
STIMULUS PRODUCTION	18	TACHYCARDIA. See PAROXYSMAL TACHYCARDIA.	
STOCK VACCINES	240	TACHYCARDIA IN NEURO-CIR- CULATORY ASTHENIA	356
"STOPPING" OF HEART	138	TACHYCARDIA OF UNDETER- MINABLE ORIGIN	142
STREPTOCOCCUS HEMOLYTICUS.	37	TECHNIQUE	
STREPTOCOCCUS VIRIDANS	37	in auscultation	70
STREPTOCOCCUS VIRIDANS AND ENDOCARDITIS	228	in blood-pressure studies..	129
STREPTOCOCCUS VIRIDANS IN MYOCARDITIS	190	in mensuration	68
STRING GALVANOMETER	104	in polygram	92
STROKING	387	in pulse study	47
STROPHANTHIN	401	TEETH	37
STROPHANTHUS	399	crowned	37
STRYCHNINE	405	devitalized	37
SUBDIAPHRAGMATIC GROWTHS.	63	infected, and endocarditis	226
SUGAR IN HEART AFFECTIONS.	211	peridental membrane	37
SUGGESTIONS IN ANALYZING ELECTROCARDIOGRAMS	123	"pulpless"	37
SUPERFICIAL CARDIAC DULL- NESS	59	pyorrhea	37
SUPRARENALIN	401	TENDERNESS	
SURGICAL ANEURISM	308	at apex of heart	45
SUSPICIOUS ATTITUDE OF MIND	416	of pectoral muscles	45
S WAVE	118	TENNIS	376
SYMPTOMS		TERMS, OBJECTIONABLE	
presenting	23	anterior axillary	67
table of	23	"cumulative action" of digi- talis	150
SYMPTOMS OF ACUTE EN- DOCARDITIS	229	"extra systoles"	136
SYMPTOMS OF MALIGNANT ENDOCARDITIS	233	"false angina"	338
SPHYGMOGRAM	91	"fatty" heart	196
SPHYGMIC PERIOD	19	"gastric flatulence"	24
SYPHILIS		heart disease	30
late appearance of heart symptoms in	36	"inherited" heart disease	40
SYPHILITIC HEART DISEASE..	193	"intercostal neuralgia"	24
"SYPHILITIC MYOCARDITIS" ..	193	mid-clavicular line	67
SYSTOLIC THRILL IN ANEU- RISM	315	mitral disease	11
		"mock angina"	338
		nipple line	67
		"pseudo angina"	338
		"rheumatism"	35
		"rheumatism of the heart".	217
		THE NITRITES	402
		THE PRE-ANEURISMAL STAGE.	309
		THERAPY, CARDIAC	381
		balneotherapy	389

	PAGE		PAGE
Therapy, Cardiac (<i>continued</i>)		Treatment of Endocarditis	236
diet	384	Treatment of Heart-block	159
exercise	383	Treatment of Intermittent	
massage	386	Claudication	290
operative procedures	388	Treatment of Pericarditis	184
rest	381	Treatment of Valvular Les-	
sanatorium treatment	388	ions	268
Thickening of Valves	244	Tricuspid Insufficiency	265
Thoracic Aneurism	312	physical signs of	265
Thrills		rarity of	264
aneurismal	44	"safety valve" type	265
carotid	44	Tricuspid Stenosis	265
pre-systolic	44, 253	physical signs of	265
significance of	44	rarity of	266
systolic, in aneurism	315	True Aneurism	307
thyroid	44	Tuberculosis	36
Thyroid	45	Tuberculosis <i>vs.</i> Heart Af-	
accessory thyroids	45	fections	361
palpation of	45	Tuberculous Pericarditis	173
Thyroid Gland in Neuro-		Tufnell's Diet	333
Circulatory Asthenia	357	Tumors, Camphorated Oil	408
Thyrototoxicosis	21	"Turning Over" of Heart	138
tachycardia of	22	T Wave	95, 120
Tidal Wave	95	T Wave of Arteriogram	95
Time Record of Polygram	91	Two to One Block	157
"To and Fro" Basal Mur-		Types	
murs	259	of cardiac patients	21
"To and Fro" Friction Rub.	168	Typhoid Fever	35
Tobacco		Typhoid Fever <i>vs.</i> Malignant	
instruction of patient con-		nant Endocarditis	235
cerning	39	"Typhoid" Form of Malignant	
statistics on use of	38	Endocarditis	233
tabagism	38	Unrestrained Activities, Ef-	
"Tobacco Heart"	138	fect of	34
Tobacco in Neuro-circula-		Uremia	291
tory Asthenia	356	Usual Standards in Blood-	
Tonicity	18	pressure studies	130
Tonicity, Diminished, Mur-		Urinalysis in Cardiac Diag-	
murs Due to	79	nosis	82
Tonsillitis	35	Use and Abuse of Electro-	
Tonsillitis and Endocar-		cardiography	125
ditis	222, 226	Uterine Life, Endocarditis	
Tracheal Tug	45	During	219
Tracing, Polygraphic	90	U Wave	121
Transient Block	156	Vaccine Therapy in Endo-	
Transmission of Murmurs	76	carditis	239
Transverse Diameter of		Valve Defects, Congenital	274
Heart	68		
Traumatic Aneurism	308		
Treatment of Aneurism	332		

	PAGE		PAGE
VALVE DEFECTS, SIGNIFICANCE OF	268	VISCERAL DISPLACEMENTS ...	65
VALVES OF THE HEART		VOLUME	
aortic	11	alternating	57
bicuspid	11	clinical terms	56
mitral	11	definition of	56
pulmonary	11	disturbances in arteriosclerosis	294
semilunar	11	irregularity of	57
VALVULAR DISEASE	242	pulsus alternans	57
aortic insufficiency	254	VON BASCH'S EMPHYSEMA...	345
stenosis	262	VON GRAEFE'S SIGN	43
congenital	276	v WAVE OF PHLEBOGRAM	95
etiology of	243		
general considerations	242	WALKING	372
incidence of	246	WANDERING WAVES IN ELECTROCARDIOGRAM	118
insufficient convalescence ..	243	WAR, EFFECT OF	354
mitral insufficiency	248	WARTHIN, STATISTICS OF	255
stenosis	249	WASSERMANN REACTION IN CARDIAC DIAGNOSIS	83
morbid anatomy	244	"WATER-HAMMER" PULSE ...	258
pulmonary insufficiency	267	WAVES	
stenosis	266	aberrant	118
purpose in diagnosing	268	a wave	95
significance of	242	c wave	95
treatment of	268	diphasic	122
tricuspid insufficiency	264	h wave	96
stenosis	266	isoelectric	120
VALVULITIS. See ENDOCARDITIS	215	jugular	95
VAGAL COMPRESSION vs. TACHYCARDIA	142	negative	121
VAGAL INFLUENCE	20	percussion	95
VAGUS, CARDIAC DISTRIBUTION OF	19	positive	121
VARIATIONS OF WAVES IN ELECTROCARDIOGRAPHY, 109, 123		split	96
VARIETIES OF ENDOCARDITIS ..	216	tidal	95
VARIETIES OF HEART-BLOCK ..	156	variations of, in electrocardiography	109, 123
VASA VASORUM	279	in jugular	96
VENESECTION IN ARTERIOSCLEROSIS	306	wandering	118
VENTRICULAR		WENCKEBACH	98
systole	15	WHAT CAN BE DONE FOR HEART DISEASE?	361
escape	162	conservation	380
flutter	162	correction	379
VENTRICULAR PREPONDERANCE. 120		early recognition	363
direction of waves in	122	patient's daily life	370
electrocardiogram of	120	prevention	366
VERRUCOSE ENDOCARDITIS	217	what cannot be done	365
VERTIGO	25	what may be done	366
VISCEROPTOSIS	64	"WHIP CORD" ARTERIES	294
		WIGGERS, CARL J.	280
		WIRING OF ANEURISM	334

	PAGE		PAGE
WITHERING, WILLIAM	394	X-RAY IN ANEURISM	324
WORDS, OBJECTIONABLE. See		X-RAY IN CARDIAC DIAG-	
"OBJECTIONABLE TERMS"		NOSIS	86
WORK AND REST, RATIO OF ..	15	X-RAY IN MYOCARDITIS	214
WRESTLING	376		
WRIST PIECE OF POLYGRAPH ..	92	ZAHN'S EMBOLISM	273



LANE MEDICAL LIBRARY

—
This book should be returned on or before
the date last stamped below.

--	--	--

L681 Smith, S.C. 80277
S65 Heart affections.
1921

[illegible]

